HEALTH ASPECTS OF PESTICIDES

abstract bulletin

March 1973 - Vol. 6, No. 3

Abstracts 73-0497-0753

HAPAB 06(03) 109-164 (1973)

Quarterly Index Issue

U.S. ENVIRONMENTAL PROTECTION AGENCY

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS

TECHNICAL SERVICES DIVISION
4770 Buford Highway, Bldg. 29
Chamblee, Georgia 30341

HEALTH ASPECTS OF PESTICIDES ABSTRACT BULLETIN is a monthly publication of the Technical Services Division of the Environmental Protection Agency.

The purpose of this publication is to foster current awareness of the major worldwide literature pertaining to the effects of pesticides on humans. The publication represents a monthly review of more than 1150 domestic and foreign journals. Subjects and authors of abstracted articles are cross-referenced in an annual index issue. Reprint requests should be directed to the author of the complete article.

Publication of an abstract, annotation, or citation does not imply endorsement by the Environmental Protection Agency of the original article or of commercial materials, equipment, or processes mentioned therein.

CONTENTS

Vol. 6 March 1973 No. 3

Page

General 109

Monitoring and Residues 118

Epidemiology, Prevention and Treatment 132

Toxicology and Pharmacology 139

Analysis 156

Quarterly Index (January-March)
- Preface I-1
- Subject Index
  - I. Compounds I-2
  - II. Concepts I-21
- Author Index
  - I. Personal I-43
  - II. Corporate I-56

HAPAB QUARTERLY INDEX

JANUARY–MARCH 1973, Vol. 6, Nos. 1-3

PREFACE

The basic design for this index was supplied by the Technical Services Division, EPA Office of Pesticide Programs. It consists of two subject indexes (compound and concept) and two author indexes (personal and corporate).

Compounds are indexed by their common names; where other compound designations are used by authors, these synonyms appear with the notation to *see* the common name. Articles referring, in general, to structural or functional chemical classes are indexed under these broader headings (e.g., organochlorines, herbicides, etc.).

Subheadings in the compound index are identical with the main headings in the concept index, the latter having a maximum of three levels of specificity in addition to the compound.

Following is a cross-referenced guide to the subject headings:

Alternative Controls
Analysis
Antidotes (*see* Treatment of Poisoning)
Associations/Organizations
Beneficial Effects
Biochemical Effects (*see also* Enzyme Activity)
Blood/Body Fluids
Carcinogenesis
Cardiovascular System
Cholinesterase (*see* Enzyme Activity)
Cholinesterase-Measurement (*see* Analysis - Enzyme-Cholinesterase)
Chromatography (*see* Analysis - Chromatography)
Conferences
Cytological Effects
Decontamination (*see* Residue Removal)
Degradation (*see* Residue Degradation *and* Metabolism)
Dermal Effects (*see* Integument)
Diet (*see* Factors Influencing Metabolism/Toxicity - Nutritional State)
Digestive System
Domestic Animals (*see* Toxicity/Domestic Animals *and* Residues/Domestic Animals)
Economics
Education/Training (*see also* Prevention)
Embryology (*see* Reproduction/Growth - Embryo/Fetus)
Endocrine System (*see also* Reproduction/Growth)
Environmental Pollution
Enzyme Activity (*see also* Biochemical Effects)
Excretory System
Experimental Animals (*see* Toxicity/Experimental Animals)
Experimental Design
Factors Influencing Metabolism/Toxicity
Food and Feed (*see* Residues/Food and Feed)
Human exposure (*see* Toxicity/Humans *and* Residues/Humans)
Immunology
Interactions (*see* Factors Influencing Metabolism/Toxicity)
Laws and Regulations (*see also* Prevention)
LD50 (*see* Toxicity/Experimental Animals)
Metabolism (*see also* Residue Degradation)
Microscopy, light and electron (*see* Cytology)
Morbidity and Mortality Statistics
Musculoskeletal System
Mutagenesis/Teratogenesis (*see also* Reproductive System)
Nervous System
New Journals
Nomenclature
Photodecomposition (*see also* Residue Degradation)
Plants (*see* Residues/Plants)
Prevention (*see also* Laws and Regulations *and* Education/Training)
Reproduction/Growth (*see also* Endocrine System *and* Mutagenesis/Teratogenesis)
Residue Degradation (*see also* Photodecomposition *and* Metabolism)
Residue Dynamics
Residue Removal
Residues/Air
Residues/Domestic Animals
Residues/Food and Feed
Residues/Human
Residues/Plants
Residues/Soil
Residues/Wildlife
Respiratory System
Reviews
Sensory System
Skeleton (*see* Musculoskeletal System)
Soil (*see* Residues/Soil)
Teratogenesis (*see* Mutagenesis/Teratogenesis)
Toxicity/Domestic Animals
Toxicity/Experimental Animals
Toxicity/Humans
Toxicity/Wildlife
Therapeutic Use
Treatment of Poisoning
Water (*see* Residues/Water)
Wildlife (*see* Residues/Wildlife *and* Toxicity/Wildlife)

Abate
see also
Organophosphates
Biochemical Effects
73-0187
Blood/Body Fluids
73-0187
Digestive System
73-0187
Endocrine System
73-0187
Enzyme activity
73-0420
Excretory System
73-0187
Metabolism
73-0420
Nervous System
73-0187
Toxicity/Experimental
Animals
73-0187
Acetophos
see Acetoxon
Acetoxon
see also
Organophosphates
Enzyme activity
73-0375
Factors Influencing
Metabolism/Toxicity
73-0375
Toxicity/Experimental
Animals
73-0375
Akar
see Chlorobenzilate
Alachlor
see also Herbicides
Analysis
73-0543
Residue Degradation
73-0543
Alar
see Aminozide
Aldicarb
see also Carbamates
Residue Removal
73-0290
Residues/Food and Feed
73-0290
Residues/Soil
73-0290
Residues/Water
73-0290
Aldrin
see also Organochlorines
Analysis
73-0204 73-0250
73-0473 73-0492
Blood/Body Fluids
73-0169
Digestive System
73-0169
Enzyme activity
73-0169
Metabolism
73-0059
Residue Degradation
73-0559
Residues/Food and Feed
72-0027 73-0595
Residues/Soil
73-0559 73-0595
Residues/Wildlife
73-0059 73-0626
Toxicity/Experimental
Animals
73-0169 73-0626
Toxicity/Wildlife
73-0626
Allethrin
see also Botanicals,
Pyrethrins
Analysis
73-0218
Factors Influencing
Metabolism/Toxicity
73-0386
Metabolism
73-0428
Nervous System
73-0386
Aluminum phosphide
see also Fumigants
Toxicity/Experimental
Animals
73-0436
Amidophos
Analysis
73-0493
Metabolism
73-0493
Aminocarb
see also Carbamates
Analysis
73-0734
**S-2-(3-Aminopropylamino)
ethyl phosphorothioic
acid**
see also
Organophosphates
Therapeutic use
73-0150
Aminozide
see also Herbicides
Residues/Food and Feed
73-0050
Amiphos
see DAEP
Anthio
see Formothion
Antidotes
see also Pralidoxime
ANTU
see also Rodenticides
Respiratory System
73-0103
Toxicity/Experimental
Animals
73-0103
Aprocarb
see Propoxur
Arsenicals
see also DSMA, MAMA,
MSMA, Neoasozin, Sodium
arsenite, Herbicides
Digestive System
73-0615
Toxicity/Humans
73-0615
Atrazine
see also Herbicides
Analysis
73-0543
Biochemical Effects
73-0097
Blood/Body Fluids
73-0097
Cardiovascular System
73-0097
Digestive System
73-0097
Endocrine System
73-0097
Enzyme activity
73-0097
Excretory System
73-0097
Factors Influencing
Metabolism/Toxicity
73-0097 73-0547
Metabolism
73-0146 73-0314
73-0547
Residue Degradation
73-0287 73-0314
73-0543 73-0547
Residues/Soil
73-0287
Respiratory System
73-0097
Toxicity/Experimental
Animals
73-0097
Avadex
see Diallate
Avicides
see 3-Chloro-p-
toluidine
Azinphosethyl
see also
Organophosphates
Analysis
73-0747
Azinphosmethyl
see also
Organophosphates
Analysis
73-0747
Metabolism
73-0415 73-0418

Toxicity/Wildlife
73-0350
Aziprotryne
see also Herbicides
Reproduction/Growth
73-0652
Toxicity/Experimental Animals
73-0652
Azodrin
see Monocrotophos
6-BAP
see also Herbicides
Digestive System
73-0185
Excretory System
73-0185
Nervous System
73-0185
Toxicity/Experimental Animals
73-0185
Barban
see also Herbicides
Toxicity/Experimental Animals
73-0430
Batestan
see Benoxaphos
Baycid
see Fenthion
Baygon
see Propoxur
Baytex
see Fenthion
Benomyl
see also Fungicides
Analysis
73-0202 73-0483
73-0725
Factors Influencing Metabolism/Toxicity
73-0419
Metabolism
73-0165 73-0183
73-0419
Reproduction/Growth
73-0165 73-0640
Residues/Soil
73-0592
Benomyl derived compounds
Analysis
73-0725
Benoxaphos
see also Organophosphates
Metabolism
73-0667
Benzimine
see also Repellents
Analysis
73-0466
Benzophosphate
see Phosalone
6-Benzylamino purine
see 6-BAP
BHC
see also Organochlorines
Analysis
73-0204 73-0210
73-0250 73-0468
Biochemical Effects
73-0396
Blood/Body Fluids
73-0446
Cytological Effects
73-0396
Digestive System
73-0083 73-0446
Enzyme activity
73-0083
Excretory System
73-0446
Integument
73-0083
Reproduction/Growth
73-0048 73-0446
Residue Removal
73-0591
Residues/Food and Feed
73-0049 73-0316
73-0575 73-0594
Residues/Humans
73-0048
Residues/Soil
73-0049
Residues/Wildlife
73-0552
Respiratory System
73-0446
Sensory System
73-0083
Toxicity/Experimental Animals
73-0396 73-0446
Toxicity/Humans
73-0083
alpha-BHC
see BHC isomers
beta-BHC
see BHC isomers
gamma-BHC
see Lindane
BHC isomers
Analysis
73-0243
Biochemical Effects
73-0392
Carcinogenesis
73-0127 73-0645
Digestive System
73-0127
Enzyme activity
73-0678
Factors Influencing Metabolism/Toxicity
73-0392 73-0678
73-0682 73-0698
Metabolism
73-0682 73-0691
73-0698
Mutagenesis/Teratogenesis
73-0691
Reproduction/Growth
73-0682 73-0691
Residue Degradation
73-0577
Residues/Food and Feed
73-0577
Residues/Soil
73-0317 73-0577
Toxicity/Experimental Animals
73-0127 73-0645
73-0682
Bioxone
see also Herbicides
Metabolism
73-0410
Bladex
see Cyanazine
Blasticidin-S
Metabolism
73-0283
Photodecomposition
73-0283
Residue Removal
73-0283
Residues/Food and Feed
73-0283
Residues/Soil
73-0283
Sensory System
73-0400 73-0659
Toxicity/Experimental Animals
73-0400 73-0659
Treatment of Poisoning
73-0659
Botanicals
see Allethrin, Furamethrin, Pyrethrins, Resmethrin
Bromophos
see also Organophosphates
Metabolism
73-0710
Residues/Air
73-0710
2-Butyl-2-ethyl-1,3-propanediol
see also Repellents
Nervous System
73-0613
Toxicity/Humans
73-0613
Treatment of Poisoning
73-0613
Captan
see also Fungicides

Analysis
73-0489
Factors Influencing Metabolism/Toxicity
73-0647
Mutagenesis/ Teratogenesis
73-0439
Reproduction/Growth
73-0439 73-0647
Toxicity/Experimental Animals
73-0439

Carbamates
see also Aldicarb, Aminocarb, Carbaryl, Carbofuran, S-2-Cyano ethyl-N-((methylcarbamoyl)oxy) thioacetimidate, DEDTC, EPTC, Meobal, Metham, Methomyl, MTMC, Propoxur, Zectran
Analysis
73-0122 73-0201
73-0233 73-0236
73-0477 73-0721
Beneficial Effects
73-0018
Environmental Pollution
73-0018
Enzyme activity
73-0084 73-0122
73-0416
Factors Influencing Metabolism/Toxicity
73-0195 73-0647
Integument
73-0084
Laws and Regulations
73-0020
Metabolism
73-0163
Morbidity and Mortality Statistics
73-0084
Reproduction/Growth
73-0647
Residues/Food and Feed
73-0020
Residues/Plants
73-0602 73-0721
Reviews
73-0020 73-0233
Sensory System
73-0084
Toxicity/Experimental Animals
73-0195 73-0699
Toxicity/Humans
73-0084

Carbaryl
see also Carbamates
Analysis
73-0216 73-0217
73-0237 73-0255
73-0484
Cytological Effects
73-0110
Digestive System
73-0196
Environmental Pollution
73-0012
Enzyme activity
73-0196
Factors Influencing Metabolism/Toxicity
73-0012 73-0367
73-0373 73-0421
73-0665
Metabolism
73-0142 73-0196
73-0367 73-0408
73-0409 73-0421
73-0434 73-0449
73-0665 73-0706
Mutagenesis/ Teratogenesis
73-0129
Nervous System
73-0196
Reproduction/Growth
73-0443
Residue Degradation
73-0665
Residue Dynamics
73-0328
Residues/Food and Feed
73-0047 73-0328
Residues/Soil
73-0044 73-0047
Toxicity/Experimental Animals
73-0129 73-0196
73-0373 73-0706

Carbaryl derived compounds
Metabolism
73-0111
Residues/Soil
73-0044

Carbofuran
see also Carbamates
Enzyme activity
73-0140
Factors Influencing Metabolism/Toxicity
73-0421
Metabolism
73-0421

Carboxide
Analysis
73-0466

Carboxin
see also Fungicides
Beneficial Effects
73-0018
Environmental Pollution
73-0018

Carbyne
see Barban

Cartap
Nervous System
73-0147
Toxicity/Experimental Animals
73-0147

Cela
see Bromophos

Celatox
see also Herbicides
Toxicity/Experimental Animals
73-0430

Ceresan M
see Granosan

Chloralose
see also Rodenticides
Metabolism
73-0707
Nervous System
73-0707
Reviews
73-0707
Toxicity/Domestic Animals
73-0707
Toxicity/Experimental Animals
73-0707
Treatment of Poisoning
73-0707

Chlorbufam
see also Herbicides
Toxicity/Experimental Animals
73-0430

Chlordane
see also Organochlorines
Analysis
73-0204 73-0224
73-0250
Biochemical Effects
73-0160 73-0184
73-0655
Digestive System
73-0160
Factors Influencing Metabolism/Toxicity
73-0184 73-0421
Metabolism
73-0160 73-0421
73-0644
Nervous System
73-0655
Residues/Soil
73-0061
Therapeutic use
73-0432
Toxicity/Experimental Animals
73-0184 73-0655

alpha-Chlordane
see Chlordane

Chlordimeform
Metabolism
73-0649
Chlorfenvinphos
see also Organophosphates
Analysis
73-0234
Enzyme activity
73-0450
Toxicity/Experimental Animals
73-0450
Chlorobenzilate
see also Organochlorines
Immunology
73-0656
2-Chloro-1-(2,4-dichlorophenyl)vinyl diethyl phosphate
see also Organophosphates
Enzyme activity
73-0143
Metabolism
73-0143
Reproduction/Growth
73-0143
Toxicity/Experimental Animals
73-0143
2-(4-Chloro-6-ethylamino-s-triazine-2-ylamino)-2-methyl propionitrile
see SD-15418
Chlorophenamidine
see Chlordimeform
3-(4-Chlorophenyl)-1-methylurea
Metabolism
73-0162
3-Chloro-p-toluidine
see also Avicides
Biochemical Effects
73-0191
Enzyme activity
73-0191
Toxicity/Experimental Animals
73-0191
Chlorpyrifos
see also Organophosphates
Analysis
73-0735
Factors Influencing Metabolism/Toxicity
73-0440
Metabolism
73-0440
Reproduction/Growth
73-0121 73-0443
Residues/Wildlife
73-0581
Toxicity/Experimental Animals
73-0440
Cobex
see Dinitramine
Compound 4072
see Chlorfenvinphos, 2-Chloro-1-(2,4-dichlorophenyl)vinyl diethyl phosphate
Copper compounds
Toxicity/Experimental Animals
73-0158
Cotoron
see Fluometuron
Coumaphos
see also Organophosphates
Enzyme activity
73-0375
Factors Influencing Metabolism/Toxicity
73-0375
Toxicity/Experimental Animals
73-0375
Crufomate
see also Organophosphates
Digestive System
73-0112
Factors Influencing Metabolism/Toxicity
73-0112
Nervous System
73-0112
Toxicity/Experimental Animals
73-0112
Cyanazine
Herbicides
73-0314 73-0590
Metabolism
73-0314 73-0590
Residue Degradation
73-0314 73-0590
S-2-Cyano ethyl-N-((methylcarbamoyl)oxy) thioacetimidate
see also Carbamates
Metabolism
73-0456
Cyprazine
see also Herbicides
Analysis
73-0543
Metabolism
73-0146
Residue Degradation
73-0543
2,4-D
see also Herbicides
Analysis
73-0722
Environmental Pollution
73-0012
Factors Influencing Metabolism/Toxicity
73-0012
Metabolism
73-0043 73-0157 73-0166
Musculoskeletal System
73-0687 73-0688
Reproduction/Growth
73-0105 73-0139
Residue Removal
73-0296
Residues/Food and Feed
73-0043 73-0157
Residues/Wildlife
73-0526
Reviews
73-0526
Sensory System
73-0086
Toxicity/Experimental Animals
73-0105
Toxicity/Humans
73-0086
Toxicity/Wildlife
73-0073 73-0526
2,4-D derived compounds
Analysis
73-0722
Digestive System
73-0186
Enzyme activity
73-0186
Toxicity/Experimental Animals
73-0186
Dacthal
see also Herbicides
Residues/Food and Feed
73-0047
Residues/Soil
73-0047
DAEP
see also Organophosphates
Blood/Body Fluids
73-0403
Cardiovascular System
73-0403
Enzyme activity
73-0403
Musculoskeletal System
73-0404
Nervous System
73-0403
Reproduction/Growth
73-0404
Respiratory System
73-0403
Toxicity/Experimental Animals
73-0403 73-0404
Dalapon
see also Herbicides

Metabolism
73-0002
Reproduction/Growth
73-0139
Reviews
73-0002

Dasanit
see Fensulfothion

2,4-DB
see also Herbicides
Reproduction/Growth
73-0093 73-0355
Toxicity/Experimental Animals
73-0355

DCNA
see Dicloran

p,p'-DDA
see DDT derived compounds

o,p'-DDD
see TDE

p,p'-DDD
see TDE

DDE
see also Organochlorines
Analysis
73-0478
Biochemical Effects
73-0170
Cytological Effects
73-0170 73-0189
Factors Influencing Metabolism/Toxicity
73-0194
Metabolism
73-0194
Reproduction/Growth
73-0339 73-0346
Residues/Air
73-0289
Residues/Food and Feed
73-0594
Residues/Humans
73-0029
Residues/Water
73-0569 73-0576
Residues/Wildlife
73-0023 73-0288
73-0292 73-0338
73-0339 73-0346
73-0478 73-0569
73-0579 73-0604
Toxicity/Experimental Animals
73-0194
Toxicity/Wildlife
73-0338 73-0339
73-0346 73-0604

DDT
see also Organochlorines
Analysis
73-0204 73-0211
73-0226 73-0230
73-0250 73-0251
73-0468 73-0469
73-0478 73-0492
73-0554 73-0715
73-0743 73-0752
Beneficial Effects
73-0011 73-0013
73-0267 73-0269
Biochemical Effects
73-0106 73-0160
73-0170 73-0180
73-0371 73-0429
73-0442 73-0655
Blood/Body Fluids
73-0190 73-0446
73-0693
Carcinogenesis
73-0452
Cytological Effects
73-0170 73-0189
73-0401 73-0661
73-0662
Digestive System
73-0083 73-0106
73-0160 73-0180
73-0442 73-0446
73-0625 73-0662
73-0693
Endocrine System
73-0117
Environmental Pollution
73-0011 73-0013
73-0267 73-0269
73-0279 73-0515
73-0535
Enzyme activity
73-0083 73-0149
73-0180 73-0368
73-0382 73-0442
73-0662
Excretory System
73-0446
Experimental Design
73-0274 73-0715
Factors Influencing Metabolism/Toxicity
73-0106 73-0117
73-0149 73-0153
73-0190 73-0194
73-0363 73-0401
73-0660 73-0672
Immunology
73-0113
Integument
73-0083
Laws and Regulations
73-0011 73-0511
73-0516 73-0517
Metabolism
73-0035 73-0152
73-0160 73-0188
73-0190 73-0194
73-0363 73-0368
73-0437 73-0660
73-0672
Morbidity and Mortality Statistics
73-0332
Nervous System
73-0371 73-0383
73-0655
Reproduction/Growth
73-0048 73-0117
73-0177 73-0192
73-0332 73-0339
73-0353 73-0359
73-0377 73-0437
73-0443 73-0446
73-0669 73-0690
73-0693
Residue Degradation
73-0544 73-0545
Residue Dynamics
73-0304
Residue Removal
73-0294 73-0296
Residues/Air
73-0289 73-0593
Residues/Domestic Animals
73-0297
Residues/Food and Feed
73-0304 73-0329
73-0545 73-0560
73-0594 73-0603
Residues/Humans
73-0029 73-0048
Residues/Soil
73-0034 73-0061
73-0304
Residues/Water
73-0035 73-0063
73-0285 73-0569
73-0593 73-0605
Residues/Wildlife
73-0023 73-0031
73-0064 73-0285
73-0288 73-0292
73-0306 73-0332
73-0338 73-0339
73-0353 73-0478
73-0552 73-0554
73-0569 73-0579
73-0626 73-0630
Respiratory System
73-0446
Reviews
73-0011 73-0013
73-0269 73-0429
73-0452
Sensory System
73-0083
Therapeutic use
73-0153 73-0429
73-0661
Toxicity/Experimental Animals
73-0113 73-0117
73-0152 73-0179
73-0180 73-0188

73-0192 73-0194
73-0359 73-0371
73-0377 73-0383
73-0446 73-0452
73-0626 73-0655
73-0662 73-0669
73-0672 73-0693
Toxicity/Humans
73-0083 73-0625
73-0675
Toxicity/Wildlife
73-0332 73-0338
73-0339 73-0353
73-0626

o,p'-DDT
see DDT isomers

DDT derived compounds
see also TDE
Cytological Effects
73-0189
Factors Influencing Metabolism/Toxicity
73-0672
Metabolism
73-0672
Residues/Water
73-0285
Residues/Wildlife
73-0285
Toxicity/Experimental Animals
73-0672

DDT isomers
Cytological Effects
73-0189

DDVP
see Dichlorvos

DEDTC
see also Carbamates
Biochemical Effects
73-0639
Digestive System
73-0639
Excretory System
73-0639

Deet
see also Repellents
Analysis
73-0466

DFP
see also Organophosphates
Biochemical Effects
73-0638
Enzyme activity
73-0167 73-0422
Factors Influencing Metabolism/Toxicity
73-0638 73-0689
Mutagenesis/Teratogenesis
73-0650
Nervous System
73-0167 73-0422
73-0426 73-0689
Reviews
73-0167
Sensory System
73-0426
Toxicity/Experimental Animals
73-0380 73-0426
73-0638 73-0650
73-0689
Treatment of Poisoning
73-0380 73-0422

Diallate
see also Herbicides
Toxicity/Experimental Animals
73-0430

Diazinon
see also Organophosphates
Metabolism
73-0059 73-0411
73-0413 73-0663
Residue Degradation
73-0663
Residues/Food and Feed
73-0026
Residues/Wildlife
73-0059 73-0581

Diazinon derived compounds
Metabolism
73-0412 73-0413

Diazoxon
see Diazinon derived compounds

Dibromochloropropane
see also Fumigants
Residue Degradation
73-0069
Residues/Water
73-0069

Dichloralurea
see also Herbicides
Analysis
73-0472

Dichlorfos
see Dichlorvos

1-(2,6-Dichlorobenzoyl)-3-(3,4-dichlorophenyl)-urea
Toxicity/Experimental Animals
73-0677

2,4-Dichloro-3'-methoxy-4'-nitro diphenyl ether
see also Herbicides
Analysis
73-0245

2,4-Dichlorophenol
see 2,4-D derived compounds

2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-oxadiazolidine-3,5-dione
see VCS-438

Dichlorvos
see also Organophosphates
Analysis
73-0252 73-0461
Cardiovascular System
73-0448
Cytological Effects
73-0096
Digestive System
73-0096 73-0381
Enzyme activity
73-0381 73-0388
73-0450 73-0681
Factors Influencing Metabolism/Toxicity
73-0081 73-0096
73-0458
Metabolism
73-0458 73-0459
Nervous System
73-0081
Prevention
73-0091
Reproduction/Growth
73-0669
Residues/Air
73-0021
Residues/Plants
73-0299
Reviews
73-0021
Toxicity/Experimental Animals
73-0096 73-0154
73-0381 73-0448
73-0450 73-0669
73-0681
Toxicity/Humans
73-0021 73-0081
73-0154
Treatment of Poisoning
73-0081 73-0448

Dicloran
see also Fungicides
Residue Degradation
73-0325

Dicofol
see also Organochlorines
Analysis
73-0211

Dieldrin
see also Organochlorines
Analysis
73-0204 73-0250
73-0492 73-0728
Biochemical Effects
73-0145 73-0160
73-0171 73-0379
73-0429
Blood/Body Fluids
73-0169
Cytological Effects
73-0360 73-0662
Digestive System
73-0104 73-0160

73-0169 73-0171
73-0662
Endocrine System
73-0379
Enzyme activity
73-0104 73-0169
73-0171 73-0662
73-0703
Factors Influencing Metabolism/Toxicity
73-0360 73-0363
73-0384 73-0423
Metabolism
73-0160 73-0188
73-0363 73-0384
73-0457 73-0562
73-0673
Nervous System
73-0171
Photodecomposition
73-0558 73-0728
Reproduction/Growth
73-0048 73-0703
Residues/Food and Feed
72-0027 73-0594
73-0595
Residues/Humans
73-0029 73-0048
Residues/Soil
73-0061 73-0595
Residues/Water
73-0569
Residues/Wildlife
73-0306 73-0562
73-0569 73-0587
73-0604 73-0626
Reviews
73-0429
Therapeutic use
73-0429
Toxicity/Experimental Animals
73-0104 73-0169
73-0171 73-0188
73-0360 73-0423
73-0626 73-0662
Toxicity/Wildlife
73-0604 73-0626

Diethamine
see Dinitramine

N,N-Diethyl-2,4-dinitro-6-trifluoromethyl-m-phenylenediamine
see Dinitramine

Diethyldithiocarbamate
see DEDTC

Diethyl toluamide
see Deet

Dimas
see Aminozide

Dimethirimol
see also Fungicides
Metabolism
73-0126

Dimethoate
see also Organophosphates
Analysis
73-0494 73-0738
Enzyme activity
73-0388
Immunology
73-0656

2,2-Dimethyl-2,3-dihydrobenzofuranyl-7-N-(dimethoxy-phosphinothioyl
Residue Degradation
73-0326

O,O-Dimethyl S-alpha-isopropoxy carbonylbenzyl phosphorodithioate
see also Organophosphates
Factors Influencing Metabolism/Toxicity
73-0141
Toxicity/Experimental Animals
73-0141

3,4-Dimethyl-N-methylcarbamate
see Meobal

Dinitramine
see also Fluorine compounds, Herbicides, Nitro compounds
Analysis
73-0724
Residue Degradation
73-0724

Dinitro-o-cresol
see also DNOC

Dinobuton
see also Nitro compounds
Factors Influencing Metabolism/Toxicity
73-0665
Metabolism
73-0665
Residue Degradation
73-0665

Dinoseb
see also Herbicides
Factors Influencing Metabolism/Toxicity
73-0665
Metabolism
73-0665
Reproduction/Growth
73-0093
Residue Degradation
73-0665
Residues/Food and Feed
73-0606

Dioxins
see also Polychlorinated biphenyls
Analysis
73-0199 73-0742
Metabolism
73-0293
Mutagenesis/Teratogenesis
73-0094
Residue Degradation
73-0293
Residues/Soil
73-0286 73-0293
Residues/Wildlife
73-0286

Dipterex
see Trichlorfon

Disulfoton
see also Organophosphates
Analysis
73-0214 73-0220
Biochemical Effects
73-0120
Factors Influencing Metabolism/Toxicity
73-0120
Metabolism
73-0680
Residue Degradation
73-0680
Residues/Food and Feed
73-0026
Residues/Soil
73-0680

Di-syston
see Disulfoton

Dithane
see Nabam

Diuron
see also Herbicides
Cardiovascular System
73-0711
Reproduction/Growth
73-0139
Residue Degradation
73-0287
Residues/Soil
73-0287 73-0321
Toxicity/Experimental Animals
73-0711

DMDT
see Methoxychlor

DNOC
see also Dinitro-o-cresol, Nitro compounds
Analysis
73-0470
Nervous System
73-0692
Reproduction/Growth
73-0093 73-0135
Respiratory System
73-0692

Toxicity/Experimental Animals
73-0692
DSMA
see also Arsenicals
Residues/Food and Feed
73-0319
Residues/Plants
73-0319
Residues/Soil
73-0319
Dursban
see Chlorpyrifos
Du-Ter
see Fentin hydroxide
Dykol 50
see DDT
EDMM
see O-Ethyl-S-(2-dimethyl-aminoethyl) methyl-thiophosphonate
Endosulfan
see also Organochlorines
Analysis
73-0253 73-0730
73-0731
Morbidity and Mortality Statistics
73-0332
Photodecomposition
73-0041
Reproduction/Growth
73-0332
Residues/Wildlife
73-0332
Toxicity/Wildlife
73-0332
Endrin
see also Organochlorines
Analysis
73-0204 73-0250
73-0492
Blood/Body Fluids
73-0671
Digestive System
73-0671
Excretory System
73-0671
Factors Influencing Metabolism/Toxicity
73-0671
Immunology
73-0113
Metabolism
73-0059
Morbidity and Mortality Statistics
73-0332
Musculoskeletal System
73-0406
Mutagenesis/Teratogenesis
73-0406
Nervous System
73-0671
Reproduction/Growth
73-0332 73-0406
Residues/Food and Feed
73-0595
Residues/Soil
73-0061 73-0595
Residues/Water
73-0569
Residues/Wildlife
73-0059 73-0332
73-0569 73-0626
Toxicity/Experimental Animals
73-0113 73-0406
73-0626 73-0671
Toxicity/Wildlife
73-0332 73-0626
EPN
see also Herbicides
Digestive System
73-0078
Enzyme activity
73-0078 73-0100
Immunology
73-0078
Integument
73-0078
Morbidity and Mortality Statistics
73-0078
Nervous System
73-0078
Toxicity/Humans
73-0078
Eptam
see EPTC
EPTC
see also Carbamates
Factors Influencing Metabolism/Toxicity
73-0134
Toxicity/Experimental Animals
73-0134
Erbitox
see DNOC
Eserine
Enzyme activity
73-0100
Estox
see also Organophosphates
Digestive System
73-0078
Enzyme activity
73-0078
Immunology
73-0078
Integument
73-0078
Morbidity and Mortality Statistics
73-0078
Nervous System
73-0078
Toxicity/Humans
73-0078
Ethylbutyl propanediol
see 2-Butyl-2-ethyl-1,3-propanediol
O-Ethyl-S-(2-dimethyl-aminoethyl) methyl-thiophosphonate
see also Organophosphates
Enzyme activity
73-0102
Nervous System
73-0102
Toxicity/Experimental Animals
73-0102
Ethylene thiourea
see Maneb derived compounds
Ethyl parathion
see Parathion
FAC
see Prothoate
Fenchlorphos
see Ronnel
Fenitrothion
see also Organophosphates
Analysis
73-0248
Biochemical Effects
73-0396
Cardiovascular System
73-0075
Cytological Effects
73-0396 73-0654
Digestive System
73-0075
Enzyme activity
73-0344 73-0375
73-0390 73-0395
73-0397 73-0679
Excretory System
73-0397
Factors Influencing Metabolism/Toxicity
73-0375 73-0397
73-0414
Metabolism
73-0114 73-0395
73-0414 73-0447
Nervous System
73-0398
Residue Dynamics
73-0045
Residues/Humans
73-0075
Sensory System
73-0390 73-0679
Toxicity/Experimental Animals
73-0375 73-0390

73-0396 73-0397
73-0398 73-0414
73-0654 73-0679
Toxicity/Humans
73-0075 73-0344

Fensulfothion
see also
Organophosphates
Analysis
73-0214 73-0723
Residue Degradation
73-0723
Residues/Soil
73-0685
Toxicity/Experimental
Animals
73-0685

Fenthion
see also
Organophosphates
Analysis
73-0244
Enzyme activity
73-0088 73-0390
73-0679
Sensory System
73-0390 73-0658
73-0679
Toxicity/Experimental
Animals
73-0390 73-0658
73-0679
Toxicity/Humans
73-0088
Treatment of Poisoning
73-0088 73-0658

Fentin
see Triphenyltin

Fentin hydroxide
see also Fungicides, Tin
compounds
Enzyme activity
73-0115
Toxicity/Experimental
Animals
73-0115

Fenuron
see also Herbicides
Endocrine System
73-0704
Excretory System
73-0704
Photodecomposition
73-0042
Toxicity/Experimental
Animals
73-0704

Fluometuron
see also Herbicides
Immunology
73-0656
Reproduction/Growth
73-0139

Fluorine compounds
see Dinitramine,
MNFA, TFM

Fluoroacetamide
see also Rodenticides
Analysis
73-0124 73-0125
73-0465
Biochemical Effects
73-0124
Blood/Body Fluids
73-0125
Digestive System
73-0125
Enzyme activity
73-0125
Nervous System
73-0402
Reproduction/Growth
73-0357
Toxicity/Experimental
Animals
73-0125 73-0357
73-0402
Treatment of Poisoning
73-0402

Formothion
see also
Organophosphates
Analysis
73-0494

Foschlor
see Trichlorfon

Fostion
see Prothoate

Fumigants
see Aluminum
phosphide,
Dibromochloropropane,
Hexachlorobutadiene,
Methyl bromide,
Phosphine

Fungicides
see also Benomyl, Captan,
Carboxin, Dicloran,
Dimethirimol, Fentin
hydroxide,
Hexachlorobenzene,
Mancozeb, Maneb,
Mercurials, Nabam,
Thiabendazole,
Thiophanate-methyl,
Thiram, Triarimol,
Tributyltin, 2,4,5-
Trichlorophenol,
Triphenyltin, Zineb
Residues/Plants
73-0546
Toxicity/Experimental
Animals
73-0158

Furamethrin
see also Botanicals,
Pyrethrins
Analysis
73-0206 73-0462

Gardona
see Chlorfenvinphos

Gramoxone
see Paraquat

Granosan
see also Mercurials
Blood/Body Fluids
73-0067
Cardiovascular System
73-0067 73-0090
73-0657
Digestive System
73-0067 73-0068
73-0090
Endocrine System
73-0068
Excretory System
73-0067
Metabolism
73-0090 73-0356
Nervous System
73-0067 73-0068
73-0090 73-0356
73-0657
Reproduction/Growth
73-0139
Residues/Air
72-0039 73-0068
Residues/Humans
73-0068
Sensory System
73-0067 73-0068
73-0356
Toxicity/Experimental
Animals
73-0356 73-0657
Toxicity/Humans
73-0067 73-0068
73-0090
Treatment of Poisoning
73-0067 73-0090

Griseofulvin
Residues/Food and Feed
73-0330

GS-13529
Metabolism
73-0146

Gusathion
see Azinphosmethyl

Guthion
see Azinphosmethyl

HCBD
see Hexachlorobutadiene

Hempa
Metabolism
73-0173

HEOD
see Dieldrin

Heptachlor
see also Organochlorines
Analysis
73-0204 73-0250
73-0492

Biochemical Effects
73-0451
Carcinogenesis
73-0197
Enzyme activity
73-0451
Metabolism
73-0059
Residues/Food and Feed
73-0303
Residues/Soil
73-0061
Residues/Wildlife
73-0059
Toxicity/Experimental Animals
73-0451

Heptachlor epoxide
see also Organochlorines
Reproduction/Growth
73-0048
Residues/Humans
73-0048
Residues/Soil
73-0061

Herbicides
see also Alachlor, Aminozide, Arsenicals, Atrazine, Aziprotryne, 6-BAP, Barban, Bioxone, Bladex, Celatox, Chlorbufam, Cyanazine, Cyprazine, 2,4-D, Dacthal, Dalapon, 2,4-DB, Diallate, Dichloralurea, 2,4-Dichloro-3'-methoxy-4'-nitro diphenyl ether, Dinitramine, Dinoseb, Diuron, EPN, Fenuron, Fluometuron, Hydroxycyprazine, Karsil, Kerb, Linuron, MAMA, MCPA, Monitor, Monuron, Oxadiazon, Paraquat, PCP, Pebulate, Picloram, Prometryne, Propanil, Pyrazon, Silvex, Simazine, Sodium chlorate, 2,4,5-T, Triazines, 2,4,5-Trichlorophenol, Trifluralin, VCS-438
Analysis
73-0482 73-0543
73-0717 73-0719
73-0737
Cardiovascular System
73-0445
Digestive System
73-0445
Excretory System
73-0445
Factors Influencing Metabolism/Toxicity
73-0373
Laws and Regulations
73-0497 73-0539
Residue Degradation
73-0543 73-0709
Residue Dynamics
73-0033
Residue Removal
73-0709
Residues/Food and Feed
73-0037
Residues/Soil
73-0037 73-0324
Reviews
73-0708
Toxicity/Experimental Animals
73-0373 73-0445
73-0709

Hexachlorane
see BHC

Hexachlorobenzene
see also Fungicides
Integument
73-0632
Toxicity/Humans
73-0632

Hexachlorobutadiene
see also Fumigants
Analysis
73-0209

Hexametapol
see Hempa

Hoe 2910
see Benoxaphos

Hydroxycyprazine
see also Herbicides
Analysis
73-0729

Iodofenphos
see also Organophosphates
Photodecomposition
73-0300

Iron methanearsonate
see Neoasozin

Karmex
see Diuron

Karsil
see also Herbicides
Metabolism
73-0646

Kelthane
see Dicofol

Kerb
see also Herbicides
Metabolism
73-0664

Kitazin-P
see also Organophosphates
Toxicity/Experimental Animals
73-0172

Lannate
see Methomyl

Lead
Environmental Pollution
73-0280

Lebaycid
see Fenthion

Legumin
see 2,4-DB

Lethane
Factors Influencing Metabolism/Toxicity
73-0414
Metabolism
73-0414
Toxicity/Experimental Animals
73-0414

Lignasan
see also Mercurials
Biochemical Effects
73-0396
Cytological Effects
73-0396
Toxicity/Experimental Animals
73-0396

Lindane
see also Organochlorines
Analysis
73-0492
Biochemical Effects
73-0160 73-0180
73-0371
Carcinogenesis
73-0645
Digestive System
73-0160 73-0180
73-0625
Enzyme activity
73-0180 73-0388
Factors Influencing Metabolism/Toxicity
72-0036 73-0682
73-0698
Integument
73-0700
Metabolism
73-0066 73-0160
73-0682 73-0694
73-0698
Nervous System
73-0066 73-0371
Reproduction/Growth
73-0048 73-0177
73-0339 73-0377
73-0391 73-0682
73-0694
Residue Degradation
73-0577
Residue Removal
73-0296
Residues/Air
73-0021
Residues/Food and Feed
72-0027 73-0305
73-0560 73-0577

Residues/Humans
73-0048
Residues/Soil
73-0034 73-0317
73-0320 73-0577
Residues/Water
72-0036 73-0063
Residues/Wildlife
73-0023 73-0288
73-0338 73-0339
Reviews
73-0021
Therapeutic use
73-0432
Toxicity/Experimental Animals
73-0179 73-0180
73-0371 73-0377
73-0645 73-0682
73-0700
Toxicity/Humans
73-0021 73-0066
73-0625
Toxicity/Wildlife
73-0338 73-0339

Linuron
see also Herbicides
Reproduction/Growth
73-0139

Malaoxon
Analysis
73-0254

Malathion
see also Organophosphates
Analysis
73-0222 73-0231
73-0248 73-0249
73-0254 73-0256
Blood/Body Fluids
73-0249
Cardiovascular System
73-0075
Cytological Effects
73-0668
Digestive System
73-0075 73-0668
Enzyme activity
73-0100 73-0138
73-0378 73-0450
73-0681
Factors Influencing Metabolism/Toxicity
73-0141 73-0378
Metabolism
73-0641
Nervous System
73-0398
Reproduction/Growth
73-0339
Residue Degradation
73-0051 73-0318
73-0563 73-0585
Residue Removal
73-0051 73-0296
Residues/Food and Feed
73-0051
Residues/Humans
73-0075
Residues/Water
73-0585
Residues/Wildlife
73-0339
Toxicity/Experimental Animals
73-0138 73-0141
73-0378 73-0398
73-0450 73-0668
73-0681
Toxicity/Humans
73-0075
Toxicity/Wildlife
73-0339 73-0350
Treatment of Poisoning
73-0378

Maleic anhydride
Laws and Regulations
73-0520

MAMA
see also Arsenicals, Herbicides
Residues/Food and Feed
73-0319
Residues/Plants
73-0319
Residues/Soil
73-0319

Mancozeb
see also Fungicides
Analysis
73-0223

Maneb
see also Fungicides
Analysis
73-0223
Endocrine System
73-0178 73-0366
Enzyme activity
73-0118
Factors Influencing Metabolism/Toxicity
73-0178 73-0366
Nervous System
73-0366
Residues/Air
73-0341
Toxicity/Experimental Animals
73-0178 73-0341
73-0366
Toxicity/Humans
73-0341

Maneb derived compounds
Analysis
73-0215 73-0221

Matacil
see Aminocarb

MBC
see Benomyl derived compounds

MCPA
see also Herbicides
Metabolism
73-0043
Residues/Food and Feed
73-0043

MCPA derived compounds
Mutagenesis/
Teratogenesis
73-0702

MCPA ethyl ester
see MCPA derived compounds

MCPEC
see MCPA derived compounds

Menazon
see also Organophosphates
Analysis
73-0471

Meobal
see also Carbamates
Enzyme activity
73-0431
Toxicity/Experimental Animals
73-0431

Mercurials
see also Granosan, Lignasan, Fungicides
Analysis
73-0475
Beneficial Effects
73-0018
Cytological Effects
73-0460
Environmental Pollution
73-0018 73-0258
73-0280 73-0536
Factors Influencing Metabolism/Toxicity
73-0647
Laws and Regulations
73-0258 73-0520
Metabolism
73-0695 73-0696
73-0697
Reproduction/Growth
73-0647 73-0690
Residues/Humans
73-0052
Residues/Water
73-0285 73-0574
Residues/Wildlife
73-0055 73-0058
73-0060 73-0285
73-0574 73-0578
73-0580
Reviews
73-0536
Toxicity/Experimental Animals
73-0460

Metaldehyde
see also Molluscicides
Nervous System
73-0174 73-0354
Respiratory System
73-0174 73-0354
Toxicity/Experimental Animals
73-0174 73-0354
Treatment of Poisoning
73-0174
Metathion
see Fenitrothion
Metham
see also Carbamates
Factors Influencing Metabolism/Toxicity
73-0547
Metabolism
73-0547
Residue Degradation
73-0547
Methomyl
see also Carbamates
Analysis
73-0239 73-0486
Digestive System
73-0078
Enzyme activity
73-0078
Immunology
73-0078
Integument
73-0078
Morbidity and Mortality Statistics
73-0078
Nervous System
73-0078
Toxicity/Humans
73-0078
Methoxychlor
see also Organochlorines
Analysis
73-0251 73-0736
Biochemical Effects
73-0160 73-0180
Digestive System
73-0160 73-0180
Enzyme activity
73-0180
Factors Influencing Metabolism/Toxicity
73-0660
Metabolism
73-0160 73-0660 73-0705
Reproduction/Growth
73-0177
Residue Degradation
73-0736
Residues/Food and Feed
73-0560
Residues/Water
73-0063
Toxicity/Experimental Animals
73-0179 73-0180 73-0705
Toxicity/Humans
73-0705
2-(Methoxy(methylthio) phosphinylimino)-3-ethyl-5-methyl-1,3-oxazolidine
Enzyme activity
73-0417
Metabolism
73-0417
Toxicity/Experimental Animals
73-0417
Methyl bromide
see also Fumigants
Biochemical Effects
73-0399
Enzyme activity
73-0399
Residue Removal
73-0298
Residues/Food and Feed
73-0561
Residues/Plants
73-0298
Toxicity/Experimental Animals
73-0399
1,1'-Methylenedi-2-naphthol
see Squoxin
Methyl ethyl thiofos
see Parathion derived compounds
Methyl mercaptophos
see also Organophosphates
Immunology
73-0656
Musculoskeletal System
73-0132
Reproduction/Growth
73-0132
Toxicity/Experimental Animals
73-0132
Methyl nitrofos
see Fenitrothion
Methylnitrophos
see Fenitrothion
Methyl parathion
see also Organophosphates
Enzyme activity
73-0088
Metabolism
73-0059
Residues/Food and Feed
73-0555 73-0603
Residues/Wildlife
73-0059
Toxicity/Humans
73-0088
Treatment of Poisoning
73-0088
Milbex
Analysis
73-0474
Milcurb
see Dimethirimol
Mirex
see also Organochlorines
Metabolism
73-0454 73-0666
Reproduction/Growth
73-0454
Toxicity/Experimental Animals
73-0454
MNFA
see also Fluorine compounds
Biochemical Effects
73-0348
Blood/Body Fluids
73-0348
Digestive System
73-0078
Enzyme activity
73-0078
Immunology
73-0078
Integument
73-0078
Morbidity and Mortality Statistics
73-0078 73-0348
Nervous System
73-0078 73-0348
Toxicity/Humans
73-0078 73-0348
Molluscicides
see Metaldehyde
Monitor
see also Herbicides
Toxicity/Experimental Animals
73-0684
Monocrotophos
see also Organophosphates
Morbidity and Mortality Statistics
73-0332
Reproduction/Growth
73-0332
Residue Degradation
73-0589
Residues/Wildlife
73-0332
Toxicity/Wildlife
73-0332
Monuron
see also Herbicides
Immunology
73-0656
Photodecomposition
73-0042

MPMC
see Meobal
MSMA
see also Arsenicals
Digestive System
73-0361
Excretory System
73-0361
Residues/Food and Feed
73-0319
Residues/Plants
73-0319
Residues/Soil
73-0319
Toxicity/Experimental Animals
73-0361
MTMC
see also Carbamates
Analysis
73-0238
M-1960
see 2-Butyl-2-ethyl-1,3-propanediol
Nabam
see also Fungicides
Digestive System
73-0078
Enzyme activity
73-0078
Immunology
73-0078
Integument
73-0078
Morbidity and Mortality Statistics
73-0078
Nervous System
73-0078
Reproduction/Growth
73-0139
Toxicity/Humans
73-0078
Naled
see also Organophosphates
Residues/Plants
73-0299
1-Naphthol
see Carbaryl derived compounds
Naptalam
Metabolism
73-0159
Nemacur
see Phenamiphos
Nemagon
see Dibromochloropropane
Nematocides
see Phenamiphos
Neoasozin
see also Arsenicals
Analysis
73-0240
Nissol
see MNFA
Nitro compounds
see also Dinitramine, Dinobuton, DNOC, PCNB
Analysis
73-0484
Toxicity/Experimental Animals
73-0158
Organochlorines
see also Aldrin, BHC, Chlordane, Chlorobenzilate, DDE, DDT, Dicofol, Dieldrin, Endosulfan, Endrin, Heptachlor, Heptachlor epoxide, Lindane, Methoxychlor, Mirex, Perthane, Tetradifon, Toxaphene
Analysis
73-0122 73-0228
73-0229 73-0232
73-0233 73-0247
73-0481 73-0485
73-0714 73-0721
73-0733 73-0745
73-0746
Conferences
73-0610
Cytological Effects
73-0460
Environmental Pollution
73-0012 73-0022
73-0501 73-0510
73-0513
Enzyme activity
73-0122 73-0633
Factors Influencing Metabolism/Toxicity
73-0012 73-0373
Laws and Regulations
73-0020 73-0520
Metabolism
73-0107 73-0502
73-0599
Musculoskeletal system
73-0609
Nervous System
73-0609
Residue Degradation
73-0570 73-0585
Residue Dynamics
73-0024
Residue Removal
73-0502 73-0549
Residues/Domestic Animals
73-0307 73-0564
Residues/Food and Feed
73-0020 73-0047
73-0062 73-0302
73-0502 73-0551
73-0553 73-0564
73-0598 73-0599
Residues/Humans
73-0053 73-0308
73-0309 73-0310
73-0311 73-0596
73-0597 73-0601
Residues/Plants
73-0602 73-0721
Residues/Soil
73-0024 73-0047
73-0571
Residues/Water
73-0022 73-0057
73-0510 73-0582
73-0584 73-0585
73-0633
Residues/Wildlife
73-0028 73-0526
73-0564 73-0565
73-0573 73-0578
73-0582 73-0614
73-0633
Reviews
73-0020 73-0022
73-0233 73-0502
73-0510 73-0526
Toxicity/Experimental Animals
73-0373 73-0460
Toxicity/Humans
73-0609 73-0610
73-0616
Toxicity/Wildlife
73-0526 73-0614
Organophosphates
see also Abate, Acetoxon, S-2-(3-Aminopropylamino) ethyl phosphorothioic acid, Azinphosethyl, Azinphosmethyl, Benoxaphos, Bromophos, Chlorfenvinphos, 2-Chloro-1-(2,4-dichlorophenyl) vinyl diethyl phosphate, Chlorpyrifos, Coumaphos, Crufomate, DAEP, DFP, Diazinon, Dichlorvos, Dimethoate, O,O-Dimethyl S-alpha-isopropoxy carbonylbenzyl phosphorodithioate, Disulfoton, Estox, O-Ethyl-S-(2-dimethyl-aminoethyl) methyl-thiophosphonate, Fenitrothion, Fensulfothion, Fenthion, Formothion, Iodofenphos, Kitazin-P, Malathion, Menazon, Methyl mercaptophos, Methyl parathion, Monocrotophos, Naled, Paraoxon, Parathion, Phenthoate, Phosalone, Phosmet,

Prothoate, Ronnel, Salithion, Sarin, Soman, Thiometon, Trichlorfon, Vamidothion
Analysis
73-0122 73-0200
73-0213 73-0233
73-0242 73-0476
73-0588 73-0721
73-0744
Biochemical Effects
73-0370
Blood/Body Fluids
73-0076
Cardiovascular System
73-0636
Conferences
73-0610
Cytological Effects
73-0460
Environmental Pollution
73-0022 73-0501
Enzyme activity
73-0076 73-0084
73-0089 73-0122
73-0342 73-0358
73-0364 73-0441
73-0622 73-0635
73-0636
Factors Influencing Metabolism/Toxicity
73-0195
Integument
73-0076 73-0084
Laws and Regulations
73-0020
Morbidity and Mortality Statistics
73-0084 73-0358
Musculoskeletal system
73-0609
Nervous System
73-0609
Prevention
73-0333
Reproduction/Growth
73-0618
Residue Dynamics
73-0024
Residue Removal
73-0549
Residues/Food and Feed
73-0020
Residues/Plants
73-0602 73-0721
Residues/Soil
73-0024 73-0588
Residues/Water
73-0022
Respiratory System
73-0636
Reviews
73-0020 73-0022
73-0233 73-0364
Sensory System
73-0076 73-0084
73-0345 73-0618
73-0620 73-0621
73-0622 73-0623
73-0624
Spectrometry
73-0476
Toxicity/Domestic Animals
73-0635
Toxicity/Experimental Animals
73-0195 73-0441
73-0460 73-0621
Toxicity/Humans
73-0076 73-0084
73-0089 73-0333
73-0342 73-0345
73-0358 73-0609
73-0610 73-0618
73-0619 73-0620
73-0621 73-0622
73-0623 73-0624
73-0636
Treatment of Poisoning
73-0089 73-0342
73-0345 73-0619
73-0631 73-0634
73-0635 73-0636

Oxadiazon
see also Herbicides
Analysis
73-0241

Paraoxon
see also Organophosphates
Biochemical Effects
73-0638
Enzyme activity
73-0422 73-0670
Factors Influencing Metabolism/Toxicity
73-0194 73-0438
73-0638 73-0670
Metabolism
73-0194 73-0438
Nervous System
73-0422
Toxicity/Experimental Animals
73-0194 73-0380
73-0638 73-0670
Treatment of Poisoning
73-0380 73-0422
73-0670

Paraquat
see also Herbicides
Beneficial Effects
73-0504
Digestive System
73-0072 73-0078
73-0347 73-0617
Enzyme activity
73-0078
Excretory System
73-0072 73-0336
73-0347 73-0617
Factors Influencing Metabolism/Toxicity
73-0081
Immunology
73-0078
Integument
73-0078
Morbidity and Mortality Statistics
73-0078 73-0617
Nervous System
73-0071 73-0078
73-0081 73-0347
Respiratory System
73-0065 73-0071
73-0072 73-0336
73-0347
Toxicity/Humans
73-0065 73-0071
73-0072 73-0078
73-0081 73-0336
73-0347 73-0617
Treatment of Poisoning
73-0065 73-0071
73-0072 73-0081
73-0336 73-0617

Parathion
see also Organophosphates
Analysis
73-0205 73-0248
73-0249
Biochemical Effects
73-0123
Blood/Body Fluids
73-0249 73-0362
Cardiovascular System
73-0075
Digestive System
73-0075
Enzyme activity
73-0092 73-0123
73-0193 73-0435
Factors Influencing Metabolism/Toxicity
73-0074 73-0194
73-0373 73-0435
Metabolism
73-0059 73-0092
73-0193 73-0194
73-0362 73-0435
Mutagenesis/ Teratogenesis
73-0405
Nervous System
73-0074 73-0637
Reproduction/Growth
73-0405
Residue Removal
73-0296

Residues/Humans
73-0075
Residues/Soil
73-0322 73-0567
Residues/Wildlife
73-0059
Therapeutic use
73-0123
Toxicity/Experimental
Animals
73-0194 73-0373
73-0405
Toxicity/Humans
73-0074 73-0075
73-0637
Treatment of Poisoning
73-0637

Parathion derived compounds
Analysis
73-0491
Enzyme activity
73-0193
Metabolism
73-0193

PCNB
see also Nitro compounds
Residue Degradation
73-0325

PCP
see also Herbicides
Analysis
73-0199 73-0479
73-0484
Biochemical Effects
73-0365
Digestive System
73-0365
Enzyme activity
73-0365
Residue Degradation
73-0284
Residues/Air
73-0313
Residues/Soil
73-0284 73-0312
Toxicity/Experimental
Animals
73-0365

Pebulate
see also Herbicides
Biochemical Effects
73-0376
Enzyme activity
73-0376
Residues/Air
73-0376
Toxicity/Experimental
Animals
73-0376

Perthane
see also Organochlorines
Analysis
73-0227

Phenamiphos
see also Nematocides
Enzyme activity
73-0686
Metabolism
73-0686
Toxicity/Experimental
Animals
73-0686

Phenthoate
see also
Organophosphates
Factors Influencing
Metabolism/Toxicity
73-0141
Toxicity/Experimental
Animals
73-0141

Phosalone
see also Organophosphates
Analysis
73-0487 73-0495
Residues/Food and Feed
73-0295

Phosmet
see also
Organophosphates
Analysis
73-0487
Laws and Regulations
73-0130
Toxicity/Experimental
Animals
73-0130

Phosphamide
see Dimethoate

Phosphamidon
Analysis
73-0740
Toxicity/Experimental
Animals
73-0335
Toxicity/Wildlife
73-0335

Phosphine
see also Fumigants
Residue Degradation
73-0566
Residues/Plants
73-0327

Phostoxin
see Aluminum phosphide

Phoxim
Residues/Soil
73-0685
Toxicity/Experimental
Animals
73-0685

Phthalaphos
see Phosmet

Picloram
see also Herbicides
Cardiovascular System
73-0137
Endocrine System
73-0137
Factors Influencing
Metabolism/Toxicity
73-0137
Residues/Soil
73-0331
Toxicity/Experimental
Animals
73-0137

Piperonyl butoxide
see also Synergists
Factors Influencing
Metabolism/Toxicity
73-0414
Metabolism
73-0414
Residues/Food and Feed
73-0315
Toxicity/Experimental
Animals
73-0414

Piscicides
see Salicylanilide I,
Squoxin

Plictran
see Tricyclohexytin
hydroxide

Polychlorinated biphenyls
see also Dioxins
Analysis
73-0226 73-0230
73-0478 73-0554
73-0743
Environmental Pollution
73-0016 73-0265
73-0278
Environmental pollution
73-0510
Environmental Pollution
73-0512 73-0534
73-0535
Prevention
73-0087
Reproduction/Growth
73-0359
Residues/Food and Feed
73-0087
Residues/Humans
73-0016 73-0311
Residues/Water
73-0510
Residues/Wildlife
73-0023 73-0064
73-0478 73-0554
73-0578 73-0579
73-0630
Reviews
73-0270 73-0278
73-0510
Toxicity/Experimental
Animals
73-0359

Pralidoxime
see also Antidotes
Metabolism
73-0151
Toxicity/Humans
73-0151
Premerge
see Dinoseb
Prometone
Biochemical Effects
73-0097
Blood/Body Fluids
73-0097
Cardiovascular System
73-0097
Digestive System
73-0097
Endocrine System
73-0097
Enzyme activity
73-0097
Excretory System
73-0097
Factors Influencing Metabolism/Toxicity
73-0097
Respiratory System
73-0097
Toxicity/Experimental Animals
73-0097
Prometryne
see also Herbicides
Factors Influencing Metabolism/Toxicity
73-0547
Metabolism
73-0547
Residue Degradation
73-0287 73-0547
Residues/Food and Feed
73-0032
Residues/Soil
73-0287
Residues/Water
73-0038
Propanil
see also Herbicides
Photodecomposition
72-0040
Propazine
Blood/Body Fluids
73-0109
Digestive System
73-0109
Endocrine System
73-0109
Immunology
73-0109 73-0352
Integument
73-0352
Metabolism
73-0146
Reproduction/Growth
73-0139
Residue Degradation
73-0323
Respiratory System
73-0109
Toxicity/Experimental Animals
73-0109 73-0430
Toxicity/Humans
73-0352
Propoxur
see also Carbamates
Analysis
73-0255 73-0726
73-0727
Digestive System
73-0181
Enzyme activity
73-0181
Metabolism
73-0449
Toxicity/Experimental Animals
73-0181
Prothoate
see also Organophosphates
Analysis
73-0741
Residues/Food and Feed
73-0603
Pyramin
see Pyrazon
Pyrazon
see also Herbicides
Toxicity/Experimental Animals
73-0430
Pyrethrins
see also Allethrin, Furamethrin, Resmethrin, Botanicals
Analysis
73-0246 73-0464
73-0480 73-0713
Immunology
73-0427
Integument
73-0334 73-0427
Metabolism
73-0428
Residues/Food and Feed
73-0315
Toxicity/Experimental Animals
73-0427
Toxicity/Humans
73-0334
Pyrocine
see Zineb
Repellents
see Benzimine, 2-Butyl-2-ethyl-1,3-propanediol, Deet
Resmethrin
see also Botanicals, Pyrethrins
Analysis
73-0207 73-0739
RH-315
see Kerb
Rodenticides
see also ANTU, Chloralose, Fluoroacetamide, Sodium fluoroacetate, Thallium, Warfarin, Zinc phosphide
Toxicity/Experimental Animals
73-0699
Ronnel
see also Organophosphates
Analysis
73-0488
Enzyme activity
73-0388 73-0433
73-0450
Metabolism
73-0107 73-0182
Toxicity/Experimental Animals
73-0433 73-0450
R-16,661
see 2-(Methoxy(methylthio) phosphinylimino)-3-ethyl-5-methyl-1,3-oxazolidine
Salicylanilide I
see also Piscicides
Residue Degradation
73-0600
Toxicity/Experimental Animals
73-0600
Salithion
see also Organophosphates
Cardiovascular System
73-0075
Digestive System
73-0075
Residues/Humans
73-0075
Toxicity/Humans
73-0075
Sarin
see also Organophosphates
Enzyme activity
73-0167 73-0369
73-0670
Factors Influencing Metabolism/Toxicity
73-0670
Nervous System
73-0167 73-0369
Reviews
73-0167

Toxicity/Experimental Animals
73-0670
Treatment of Poisoning
73-0369 73-0670

Sayphos
see Menazon

SD-15418
Analysis
73-0543
Residue Degradation
73-0543

Select
see Naptalam

Sevin
see Carbaryl

Silvex
see also Herbicides
Metabolism
73-0043 73-0157
Residues/Food and Feed
73-0043 73-0157

Simazine
see also Herbicides
Factors Influencing
Metabolism/Toxicity
73-0547
Immunology
73-0352
Integument
73-0352
Metabolism
73-0146 73-0314
73-0547
Residue Degradation
73-0314 73-0547
Toxicity/Humans
73-0352

Sodium arsenite
see also Arsenicals
Residues/Food and Feed
73-0054
Residues/Soil
73-0054

Sodium chlorate
see also Herbicides
Biochemical Effects
73-0176
Residue Degradation
73-0175
Residues/Plants
73-0175
Residues/Soil
73-0175
Toxicity/Experimental Animals
73-0175 73-0176

Sodium fluoroacetate
see also Rodenticides
Biochemical Effects
73-0385
Toxicity/Experimental Animals
73-0385

Soman
see also Organophosphates
Enzyme activity
73-0167
Nervous System
73-0167
Reviews
73-0167

Squoxin
see also Piscicides
Factors Influencing Metabolism/Toxicity
73-0425
Toxicity/Experimental Animals
73-0425

Sumithion
see Fenitrothion

Synergists
see also Piperonyl butoxide
Analysis
73-0480

2,4,5-T
see also Herbicides
Environmental Pollution
73-0017
Laws and Regulations
73-0017 73-0520
Metabolism
73-0043 73-0157
Mutagenesis/Teratogenesis
73-0085 73-0119
Prevention
73-0085
Reproduction/Growth
73-0017 73-0085
73-0119
Residues/Food and Feed
73-0043 73-0157
Residues/Soil
73-0312

Tamaron
see Monitor

TBZ
see Thiabendazole

TCA
Residues/Food and Feed
73-0557
Residues/Plants
73-0557

TCDD
see Dioxins

TDE
see also Organochlorines
Analysis
73-0251 73-0478
73-0752
Biochemical Effects
73-0424
Carcinogenesis
73-0108
Cytological Effects
73-0189 73-0661
Digestive System
73-0155 73-0651
Endocrine System
73-0108 73-0424
Factors Influencing Metabolism/Toxicity
73-0363 73-0672
Immunology
73-0651
Metabolism
73-0363 73-0672
Reproduction/Growth
73-0108
Residues/Food and Feed
73-0594
Residues/Water
73-0569
Residues/Wildlife
73-0292 73-0478
73-0569 73-0587
Reviews
73-0108
Therapeutic use
73-0155 73-0651
73-0661
Toxicity/Experimental Animals
73-0108 73-0424
73-0672
Toxicity/Humans
73-0155

Tedion
see Tetradifon

2,3,7,8-Tetrachloro dibenzo-p-dioxin
see Dioxins

Tetradifon
see also Organochlorines
Enzyme activity
73-0387

TFM
see also Fluorine compounds
Metabolism
73-0116

Thallium
see also Rodenticides
Cardiovascular System
73-0628
Integument
73-0612 73-0628
Metabolism
73-0612
Nervous System
73-0612 73-0628
Toxicity/Humans
73-0612 73-0627
73-0628
Treatment of Poisoning
73-0627 73-0628

Thanite
Factors Influencing

Metabolism/Toxicity
73-0414
Metabolism
73-0414
Toxicity/Experimental Animals
73-0414
Thiabendazole
see also Fungicides
Analysis
73-0732
Metabolism
73-0164
Residues/Food and Feed
73-0568
Thiodan
see Endosulfan
Thiometon
see also Organophosphates
Analysis
73-0490
Residues/Food and Feed
73-0490
Thiophanate
Analysis
73-0483
Blood/Body Fluids
73-0407
Cardiovascular System
73-0407
Digestive System
73-0407
Endocrine System
73-0407
Excretory System
73-0407
Factors Influencing Metabolism/Toxicity
73-0419
Metabolism
73-0419
Mutagenesis/ Teratogenesis
73-0148
Nervous System
73-0407
Respiratory System
73-0407
Toxicity/Experimental Animals
73-0148 73-0407
Thiophanate-methyl
see also Fungicides
Analysis
73-0483
Mutagenesis/ Teratogenesis
73-0148
Toxicity/Experimental Animals
73-0148
Thio-TEPA
Cytological Effects
73-0372
Factors Influencing Metabolism/Toxicity
73-0372
Mutagenesis/ Teratogenesis
73-0372
Thiram
see also Fungicides
Biochemical Effects
73-0444
Digestive System
73-0068 73-0629
Endocrine System
73-0068
Enzyme activity
73-0629
Factors Influencing Metabolism/Toxicity
73-0547 73-0629
Metabolism
73-0547
Nervous System
73-0068
Residue Degradation
73-0547
Residues/Air
73-0068
Residues/Humans
73-0068
Sensory System
73-0068
Toxicity/Experimental Animals
73-0444
Toxicity/Humans
73-0068 73-0629
Treatment of Poisoning
73-0629
Thiuram
see Thiram
Tillam
see Pebulate
Tin compounds
see Fentin hydroxide, Tributyltin, Tricyclohexytin hydroxide
TMTD
see Thiram
Topsin
see Thiophanate
Topsin-M
see Thiophanate-methyl
Tordon-22K
see Picloram
Toxaphene
see also Organochlorines
Analysis
73-0205 73-0716
Biochemical Effects
73-0160
Cardiovascular System
73-0683
Cytological Effects
73-0683
Digestive System
73-0160
Endocrine System
73-0453
Metabolism
73-0160
Morbidity and Mortality Statistics
73-0332
Reproduction/Growth
73-0332
Residues/Soil
73-0061
Residues/Water
73-0056
Residues/Wildlife
73-0332
Reviews
73-0716
Toxicity/Experimental Animals
73-0453 73-0683
Toxicity/Wildlife
73-0332
Triamiphos
Biochemical Effects
73-0123
Enzyme activity
73-0123
Therapeutic use
73-0123
Triarimol
see also Fungicides
Metabolism
73-0161
Toxicity/Experimental Animals
73-0161
Triazines
see also Herbicides
Analysis
73-0225
Tributyltin
see also Fungicides, Tin compounds
Photodecomposition
73-0572
Trichlorfon
see also Organophosphates
Analysis
73-0212 73-0461
73-0720
Blood/Body Fluids
73-0351 73-0446
Cardiovascular System
73-0448
Digestive System
73-0112 73-0351
73-0446
Enzyme activity
73-0156 73-0450
73-0681
Excretory System
73-0446

Factors Influencing Metabolism/Toxicity
73-0074 73-0112
Immunology
73-0136
Nervous System
73-0074 73-0112
Reproduction/Growth
73-0446
Residues/Air
73-0712
Residues/Food and Feed
73-0603
Residues/Plants
73-0299
Respiratory System
73-0351 73-0446
Toxicity/Experimental Animals
73-0112 73-0136
73-0156 73-0158
73-0380 73-0446
73-0448 73-0450
73-0681 73-0712
Toxicity/Humans
73-0074 73-0351
Treatment of Poisoning
73-0156 73-0351
73-0380 73-0448

Trichlormetaphos-3
see Ronnel

2,4,5-Trichlorophenol
see also Fungicides, Herbicides
Immunology
73-0349
Integument
73-0349
Toxicity/Humans
73-0349

Tricyclohexytin hydroxide
see also Tin compounds
Enzyme activity
73-0115
Toxicity/Experimental Animals
73-0115

3-Trifluoromethyl-4-nitrophenol
see TFM

Trifluralin
see also Herbicides
Residue Degradation
73-0287
Residues/Food and Feed
73-0047
Residues/Soil
73-0047 73-0287

Triphenyltin
see also Fungicides
Metabolism
73-0676
Photodecomposition
73-0572
Residues/Food and Feed
73-0030
Residues/Soil
73-0030

Tritox
see DDT, Lindane, Methoxychlor

USB 3584
see Dinitramine

Vamidothion
see also Organophosphates
Metabolism
73-0101

Vapam
see Metham

Vapona
see Dichlorvos

VCS-438
see also Herbicides
Metabolism
73-0144

Warfarin
see also Rodenticides
Toxicity/Domestic Animals
73-0608
Treatment of Poisoning
73-0608

Weedol
see Paraquat

Wofatox
see Methyl parathion

WR-2721
see S-2-(3-Aminopropylamino)ethyl phosphorothioic acid

3,5-Xylyl methylcarbamate
Residues/Soil
73-0044

X-52
see 2,4-Dichloro-3'-methoxy-4'-nitro diphenyl ether

Zectran
see also Carbamates
Analysis
73-0734

Zinc phosphide
see also Rodenticides
Residue Degradation
73-0566

Zineb
see also Fungicides
Analysis
73-0208 73-0223
Cytological Effects
73-0133 73-0654
Endocrine System
73-0178
Enzyme activity
73-0374
Excretory System
73-0133
Factors Influencing Metabolism/Toxicity
73-0178
Musculoskeletal System
73-0653
Mutagenesis/Teratogenesis
73-0653
Residues/Air
73-0374
Toxicity/Experimental Animals
73-0133 73-0178
73-0374 73-0653
73-0654

Ziram
Cytological Effects
73-0099 73-0128
73-0133
Enzyme activity
73-0099
Excretory System
73-0133
Integument
73-0070
Metabolism
73-0131
Mutagenesis/Teratogenesis
73-0128
Prevention
73-0070
Residues/Air
73-0070
Respiratory System
73-0070
Sensory System
73-0070
Toxicity/Experimental Animals
73-0133

Alternative Controls
73-0010 73-0257
73-0259 73-0276
73-0393 73-0394
73-0508 73-0509
73-0521 73-0527
73-0529

Analysis
General
73-0748
BHC isomers
73-0243
Carbamates
73-0233
DDT
73-0715
Disulfoton
73-0220
Organochlorines
73-0233 73-0481
Organophosphates
73-0233
Toxaphene
73-0716
Bioassay
Alachlor
73-0543
Atrazine
73-0543
Cyprazine
73-0543
Fluoroacetamide
73-0124 73-0125
Herbicides
73-0543
Ronnel
73-0488
SD-15418
73-0543
Chromatography
General
Aldrin
73-0204 73-0492
BHC
73-0204
Chlordane
73-0204
DDT
73-0204 73-0492
73-0554
Dieldrin
73-0204 73-0492
Endrin
73-0204 73-0492
Heptachlor
73-0204 73-0492
Lindane
73-0492
Maneb derived
compounds
73-0221
Parathion
73-0205
Polychlorinated
biphenyls
73-0554
Toxaphene
73-0205
Column
DDE
73-0478
DDT
73-0226 73-0230
73-0478 73-0752
Dichloralurea
73-0472
Organochlorines
73-0228
Parathion derived
compounds
73-0491
Polychlorinated
biphenyls
73-0226 73-0230
73-0478
TDE
73-0478 73-0752
Electrophoresis
Trichlorfon
73-0720
Gas-liquid
73-0198 73-0718
73-0751
Allethrin
73-0218
Carbamates
73-0236 73-0721
Carbaryl
73-0217 73-0237
73-0484
Chlordane
73-0224
Chlorpyrifos
73-0735
DDT
73-0743 73-0752
2,4-Dichloro-3'-
methoxy-4'-nitro
diphenyl ether
73-0245
Dieldrin
73-0728
Dimethoate
73-0738
Dinitramine
73-0724
Dioxins
73-0199 73-0742
Endosulfan
73-0730 73-0731
Fensulfothion
73-0723
Furamethrin
73-0206 73-0462
Hydroxycyprazine
73-0729
Maneb derived
compounds
73-0215
Methomyl
73-0239
MTMC
73-0238
Nitro compounds
73-0484
Organochlorines
73-0228 73-0229
73-0232 73-0485
73-0721 73-0733
Organophosphates
73-0242 73-0721
PCP
73-0199 73-0479
73-0484
Perthane
73-0227
Phosphamidon
73-0740
Polychlorinated
biphenyls
73-0743
Propoxur
73-0726 73-0727
Prothoate
73-0741
Pyrethrins
73-0480 73-0713
Resmethrin
73-0207 73-0739
Synergists
73-0480
TDE
73-0752
Thiometon
73-0490
Triazines
73-0225
Liquid-liquid
Methomyl
73-0486
Paper
Trichlorfon
73-0212
Thin-layer
Aldrin
73-0250 73-0473
Amidophos
73-0493
Aminocarb
73-0734
Azinphosethyl
73-0747
Azinphosmethyl
73-0747
Benomyl
73-0483 73-0725
Benomyl derived
compounds
73-0725

Benzimine
73-0466
BHC
73-0210 73-0250
73-0468
Carbamates
73-0477
Carbaryl
73-0216
Carboxide
73-0466
Chlordane
73-0250
Chlorfenvinphos
73-0234
2,4-D
73-0722
2,4-D derived compounds
73-0722
DDT
73-0250 73-0251
73-0468
Deet
73-0466
Dichlorvos
73-0252
Dieldrin
73-0250
Dimethoate
73-0494
Endrin
73-0250
Fenitrothion
73-0248
Fenthion
73-0244
Formothion
73-0494
Heptachlor
73-0250
Herbicides
73-0717 73-0719
Malaoxon
73-0254
Malathion
73-0248 73-0249
73-0254
Methoxychlor
73-0251 73-0736
Organochlorines
73-0247
Organophosphates
73-0476 73-0588
73-0744
Oxadiazon
73-0241
Parathion
73-0248 73-0249
Pyrethrins
73-0246
TDE
73-0251
Thiabendazole
73-0732
Thiophanate
73-0483
Thiophanate-methyl
73-0483
Zectran
73-0734
Electrometry
Polarography
DNOC
73-0470
Menazon
73-0471
Organophosphates
73-0213
Trichlorfon
73-0720
Enzyme
Carbamates
73-0122
Organochlorines
73-0122
Organophosphates
73-0122
General
Herbicides
73-0482
Aconitase
Fluoroacetamide
73-0465
Cholinesterase
73-0203 73-0463
73-0496 73-0749
73-0750 73-0753
Carbamates
73-0122
Carbaryl
73-0255
Chlorfenvinphos
73-0234
Dichlorvos
73-0252
Malaoxon
73-0254
Malathion
73-0254
Organochlorines
73-0122
Organophosphates
73-0122 73-0744
Propoxur
73-0255
Ronnel
73-0488
Spectrometry
General
Dieldrin
73-0728
Colorimetry
Azinphosethyl
73-0747
Azinphosmethyl
73-0747
Captan
73-0489
DDT
73-0211
Dicofol
73-0211
Disulfoton
73-0214
Endosulfan
73-0253
Fensulfothion
73-0214
Herbicides
73-0717 73-0737
Malathion
73-0222 73-0256
Mancozeb
73-0223
Maneb
73-0223
Milbex
73-0474
Neoasozin
73-0240
Organochlorines
73-0247
Phosalone
73-0495
Pyrethrins
73-0246 73-0464
Zineb
73-0208 73-0223
Fluorometry
Aminocarb
73-0734
Carbamates
73-0201
Dichlorvos
73-0461
Organophosphates
73-0476
Thiabendazole
73-0732
Trichlorfon
73-0461
Zectran
73-0734
Infrared
73-0219
Chlordane
73-0224
Maneb derived
compounds
73-0221
Methomyl
73-0239
Mass spectrometry
73-0751
Carbamates
73-0477
Carbaryl
73-0217
Maneb derived
compounds
73-0215 73-0221
Mercurials
73-0475

Organochlorines
73-0714 73-0745
73-0746
UV
Benomyl
73-0202
DDT
73-0469
Fenthion
73-0244
Hexachlorobutadiene
73-0209
Organophosphates
73-0200
Titration
Malathion
73-0231
Phosalone
73-0487
Phosmet
73-0487
Beneficial Effects
73-0010 73-0263
73-0276 73-0281
73-0537 73-0540
Carbamates
73-0018
Carboxin
73-0018
DDT
73-0011 73-0013
73-0267 73-0269
Mercurials
73-0018
Paraquat
73-0504
Biochemical Effects
General
BHC isomers
73-0392
Dieldrin
73-0379
Animals/Experimental
Chlordane
73-0655
DDE
73-0170
DDT
73-0170 73-0655
Heptachlor
73-0451
Pebulate
73-0376
Human
MNFA
73-0348
Plant
DDE
73-0170
DDT
73-0170
Amino acids/peptides/proteins
Animals/Experimental
Atrazine
73-0097
Chlordane
73-0184
DDT
73-0371
Lindane
73-0371
Prometone
73-0097
Sodium fluoroacetate
73-0385
Carbohydrates
Animals/Experimental
3-Chloro-p-toluidine
73-0191
DDT
73-0180
Lindane
73-0180
Methoxychlor
73-0180
Catecholamines
Animals/Experimental
DFP
73-0638
Disulfoton
73-0120
Paraoxon
73-0638
Citric acid
Animals/Experimental
Fluoroacetamide
73-0124
Electrolytes
Animals/Experimental
DDT
73-0442
Lipids/steroids/sterols
Animals/Experimental
Abate
73-0187
BHC
73-0396
Dieldrin
73-0145 73-0379
Fenitrothion
73-0396
Lignasan
73-0396
Parathion
73-0123
PCP
73-0365
TDE
73-0424
Triamiphos
73-0123
Nucleic Acids
Animals/Experimental
DDT
73-0180
Dieldrin
73-0171
Lindane
73-0180
Methoxychlor
73-0180
Thiram
73-0444
In vitro
Organophosphates
73-0370
Porphyrins
Animals/Experimental
DDT
73-0429
Dieldrin
73-0429
Sodium chlorate
73-0176
Respiration
In vitro
DEDTC
73-0639
Vitamins/coenzymes
Animals/Experimental
Chlordane
73-0160
DDT
73-0106 73-0160
Dieldrin
73-0160
Lindane
73-0160
Methoxychlor
73-0160
Methyl bromide
73-0399
Toxaphene
73-0160
Blood/Body Fluids
General
DAEP
73-0403
Malathion
73-0249
Parathion
73-0249
Animals/Experimental
Endrin
73-0671
Propazine
73-0109
Thiophanate
73-0407
Blood-brain barrier
Animals/Experimental
Parathion
73-0362
In vitro
DDT
73-0190
Cells
Animals/Experimental
Abate
73-0187
BHC
73-0446

DDT
73-0446 73-0693
Trichlorfon
73-0446
Human
73-0001 73-0077
Granosan
73-0067
MNFA
73-0348
Organophosphates
73-0076
Trichlorfon
73-0351
Plasma/serum
Animals/Experimental
Aldrin
73-0169
Atrazine
73-0097
Dieldrin
73-0169
Fluoroacetamide
73-0125
Prometone
73-0097
Carcinogenesis
General
73-0519
Animals/experimental
73-0389
BHC isomers
73-0127 73-0645
DDT
73-0452
Heptachlor
73-0197
Lindane
73-0645
TDE
73-0108
Cardiovascular System
General
Animals/Experimental
DAEP
73-0403
Granosan
73-0657
Human
Granosan
73-0067
Blood vessels
Animals/Experimental
Herbicides
73-0445
Toxaphene
73-0683
Heart
Animals/Experimental
Atrazine
73-0097
Dichlorvos
73-0448
Diuron
73-0711
Picloram
73-0137
Prometone
73-0097
Thiophanate
73-0407
Trichlorfon
73-0448
Human
Fenitrothion
73-0075
Granosan
73-0090
Malathion
73-0075
Organophosphates
73-0636
Parathion
73-0075
Salithion
73-0075
Thallium
73-0628
Conferences
73-0340
Organochlorines
73-0610
Organophosphates
73-0610
Cytological Effects
General
Animals/Experimental
DDE
73-0170
DDT
73-0170
Mercurials
73-0460
Organochlorines
73-0460
Organophosphates
73-0460
Toxaphene
73-0683
In vitro
DDT
73-0661
Fenitrothion
73-0654
TDE
73-0661
Zineb
73-0654
Plant
DDE
73-0170
DDT
73-0170
Chromosomes/genes
In vitro
DDE
73-0189
DDT
73-0189
DDT derived
compounds
73-0189
DDT isomers
73-0189
TDE
73-0189
Thio-TEPA
73-0372
Ziram
73-0128
Endoplasmic reticulum
Animals/Experimental
Dichlorvos
73-0096
Growth
Plant
DDT
73-0401
Microsomes
Animals/Experimental
DDT
73-0662
Dieldrin
73-0662
Malathion
73-0668
Mitochondria
Microorganisms
Ziram
73-0099
Mitosis/meiosis
In vitro
Carbaryl
73-0110
DDE
73-0189
DDT
73-0189
DDT derived
compounds
73-0189
DDT isomers
73-0189
TDE
73-0189
Zineb
73-0133
Ziram
73-0133
Morphology
Animals/Experimental
BHC
73-0396
Dieldrin
73-0360
Fenitrothion
73-0396
Lignasan
73-0396
Digestive System
General
Animals/Experimental
Atrazine
73-0097

Dichlorvos
73-0381
Endrin
73-0671
Herbicides
73-0445
Prometone
73-0097
Propazine
73-0109
Thiophanate
73-0407
Human
EPN
73-0078
Estox
73-0078
Granosan
73-0067 73-0090
Methomyl
73-0078
MNPA
73-0078
Nabam
73-0078
Paraquat
73-0078
TDE
73-0651
Thiram
73-0629
Trichlorfon
73-0351
Alimentary Tract
Animals/Experimental
BHC
73-0446
Crufomate
73-0112
DDT
73-0446
MSMA
73-0361
Trichlorfon
73-0112 73-0446
Human
TDE
73-0155
Liver
BHC
73-0446
DDT
73-0446
Trichlorfon
73-0446
Animals/Experimental
Abate
73-0187
Aldrin
73-0169
BHC isomers
73-0127
Carbaryl
73-0196
Chlordane
73-0160
2,4-D derived compounds
73-0186
DDT
73-0106 73-0160
73-0180 73-0442
73-0662 73-0693
Dichlorvos
73-0096
Dieldrin
73-0104 73-0160
73-0169 73-0171
73-0662
Fluoroacetamide
73-0125
Lindane
73-0160 73-0180
Malathion
73-0668
Methoxychlor
73-0160 73-0180
MSMA
73-0361
PCP
73-0365
Propoxur
73-0181
Toxaphene
73-0160
Human
73-0077
Arsenicals
73-0615
BHC
73-0083
DDT
73-0083 73-0625
Fenitrothion
73-0075
Granosan
73-0068
Lindane
73-0625
Malathion
73-0075
Paraquat
73-0072 73-0347
73-0617
Parathion
73-0075
Salithion
73-0075
Thiram
73-0068
In vitro
DEDTC
73-0639
Pancreas
Animals/Experimental
6-BAP
73-0185
DDT
73-0442
Economics
73-0008 73-0275
73-0498 73-0499
73-0518 73-0529
73-0541
Education/Training
73-0260
Endocrine System
General
Animals/Experimental
Fenuron
73-0704
Toxaphene
73-0453
Adrenal
Animals/Experimental
Abate
73-0187
Atrazine
73-0097
Dieldrin
73-0379
Maneb
73-0366
Prometone
73-0097
TDE
73-0108 73-0424
Pituitary
Animals/Experimental
DDT
73-0117
Spleen
Animals/Experimental
Picloram
73-0137
Propazine
73-0109
Thyroid
Animals/Experimental
Atrazine
73-0097
Maneb
73-0178
Prometone
73-0097
Thiophanate
73-0407
Zineb
73-0178
Human
Granosan
73-0068
Thiram
73-0068
Environmental Pollution
73-0001 73-0004
73-0005 73-0007
73-0009 73-0010
73-0014 73-0015
73-0259 73-0263
73-0264 73-0268
73-0271 73-0281
73-0282 73-0498

73-0499 73-0500
73-0505 73-0514
73-0521 73-0522
73-0524 73-0525
73-0528 73-0530
73-0531 73-0537
73-0540
Carbamates
73-0018
Carbaryl
73-0012
Carboxin
73-0018
2,4-D
73-0012
DDT
73-0011 73-0013
73-0267 73-0269
73-0279 73-0515
73-0535
Lead
73-0280
Mercurials
73-0018 73-0258
73-0280 73-0536
Organochlorines
73-0012 73-0022
73-0501 73-0510
73-0513
Organophosphates
73-0022 73-0501
Polychlorinated biphenyls
73-0016 73-0265
73-0278 73-0510
73-0512 73-0534
73-0535
2,4,5-T
73-0017
Enzyme activity
General
73-0082 73-0674
Aldrin
73-0169
Atrazine
73-0097
BHC isomers
73-0678
Carbamates
73-0084
3-Chloro-p-toluidine
73-0191
DDT
73-0368
Dichlorvos
73-0681
Dieldrin
73-0104 73-0169
73-0171 73-0703
Fluoroacetamide
73-0125
Malathion
73-0681
Organophosphates
73-0076 73-0084
Pebulate
73-0376
Prometone
73-0097
Propoxur
73-0181
Trichlorfon
73-0681
Ziram
73-0099
Alkaline phosphatase
Dichlorvos
73-0388
Dimethoate
73-0388
Lindane
73-0388
Ronnel
73-0388
Aminopyrine demethylase
Carbaryl
73-0196
Aniline hydroxylase
Carbaryl
73-0196
Aryl hydrocarbon hydroxylase
DDT
73-0149
ATPase
DDT
73-0382 73-0442
Fentin hydroxide
73-0115
Tetradifon
73-0387
Tricyclohexytin hydroxide
73-0115
Beta-Glucuronidase
Methyl bromide
73-0399
Cholinesterase
73-0496
Abate
73-0420
Acetoxon
73-0375
BHC
73-0083
Carbamates
73-0122 73-0415
Carbofuran
73-0140
Chlorfenvinphos
73-0450
2-Chloro-1-(2,4-dichlorophenyl) vinyl diethyl phosph
73-0143
Coumaphos
73-0375
DAEP
73-0403
DDT
73-0083
DFP
73-0167 73-0422
Dichlorvos
73-0381 73-0450
EPN
73-0078 73-0100
Eserine
73-0100
Estox
73-0078
O-Ethyl-S-(2-dimethyl-aminoethyl) methyl-thiophosphona
73-0102
Fenitrothion
73-0344 73-0375
73-0390 73-0395
73-0397 73-0679
Fenthion
73-0088 73-0390
73-0679
Heptachlor
73-0451
Malathion
73-0100 73-0138
73-0378 73-0450
Meobal
73-0431
Methomyl
73-0078
2-(Methoxy(methylthio) phosphinylimino)-3-e
73-0417
Methyl parathion
73-0088
MNFA
73-0078
Nabam
73-0078
Organochlorines
73-0122 73-0633
Organophosphates
73-0089 73-0122
73-0342 73-0358
73-0364 73-0441
73-0622 73-0635
73-0636
Paraoxon
73-0422 73-0670
Paraquat
73-0078
Parathion
73-0123
Phenamiphos
73-0686
Ronnel
73-0433 73-0450
Sarin
73-0167 73-0369
73-0670
Soman
73-0167

Thiram
73-0629
Triamiphos
73-0123
Trichlorfon
73-0156 73-0450
Zineb
73-0374
Cytochrome oxidase
DDT
73-0180
Lindane
73-0180
Methoxychlor
73-0180
GPT
2,4-D derived compounds
73-0186
PCP
73-0365
Lactic dehydrogenase
Maneb
73-0118
Mixed function oxidases
DDT
73-0662
Dieldrin
73-0662
Parathion
73-0092 73-0193
73-0435
Parathion derived compounds
73-0193
Peroxidase
Dichlorvos
73-0388
Dimethoate
73-0388
Lindane
73-0388
Ronnel
73-0388
Succinic dehydrogenase
DDT
73-0180
Lindane
73-0180
Methoxychlor
73-0180
Excretory System
General
Animals/Experimental
Atrazine
73-0097
Fenuron
73-0704
Prometone
73-0097
Thiophanate
73-0407
Kidney
Animals/Experimental
Abate
73-0187
6-BAP
73-0185
BHC
73-0446
DDT
73-0446
Endrin
73-0671
Fenitrothion
73-0397
Herbicides
73-0445
MSMA
73-0361
Trichlorfon
73-0446
Human
Granosan
73-0067
Paraquat
73-0072 73-0336
73-0347 73-0617
In vitro
DEDTC
73-0639
Zineb
73-0133
Ziram
73-0133
Experimental Design
73-0019 73-0264
73-0271 73-0272
73-0389 73-0542
73-0583
DDT
73-0274 73-0715
Factors Influencing Metabolism/Toxicity
General
73-0643
Atrazine
73-0097
Prometone
73-0097
Adaptation
Carbaryl
73-0012
2,4-D
73-0012
DDT
73-0190 73-0401
DFP
73-0689
Organochlorines
73-0012
Age
Carbamates
73-0195
Maneb
73-0366
Organophosphates
73-0195
Biological magnification
73-0611
DDT
73-0660
Dieldrin
73-0384
Methoxychlor
73-0660
Diet
73-0343
Carbaryl
73-0373
Herbicides
73-0373
Organochlorines
73-0373
Parathion
73-0373
Disease state
Dichlorvos
73-0081
Paraquat
73-0081
Squoxin
73-0425
Interactions
73-0095
Acetoxon
73-0375
Atrazine
73-0547
BHC isomers
73-0392 73-0678
Carbaryl
73-0421 73-0665
Carbofuran
73-0421
Chlordane
73-0184 73-0421
Coumaphos
73-0375
DDE
73-0194
DDT
73-0106 73-0149
73-0153 73-0194
73-0363
DFP
73-0638
Dichlorvos
73-0096
Dieldrin
73-0360 73-0363
73-0423
O,O-Dimethyl S-alpha-isopropoxy carbonylbenzyl phosp
73-0141
Dinobuton
73-0665
Dinoseb
73-0665
Fenitrothion
73-0375 73-0414

Lethane
73-0414
Malathion
73-0141 73-0378
Maneb
73-0178
Metham
73-0547
Paraoxon
73-0194 73-0438
73-0638 73-0670
Parathion
73-0074 73-0194
73-0435
Phenthoate
73-0141
Piperonyl butoxide
73-0414
Prometryne
73-0547
Sarin
73-0670
Simazine
73-0547
TDE
73-0363
Thanite
73-0414
Thio-TEPA
73-0372
Thiram
73-0547 73-0629
Trichlorfon
73-0074
Zineb
73-0178
Nutritional state
BHC isomers
73-0682 73-0698
Carbaryl
73-0367
DDT
73-0672
DDT derived compounds
73-0672
Lindane
73-0682 73-0698
TDE
73-0672
pH
Benomyl
73-0419
Thiophanate
73-0419
Route
Dichlorvos
73-0458
Schedule of dosage
Crufomate
73-0112
DDT
73-0117
Endrin
73-0671
Trichlorfon
73-0112
Sex
73-0077
Dieldrin
73-0360
Disulfoton
73-0120
Solubility
Lindane
72-0036
Species
Captan
73-0647
Carbamates
73-0647
Carbaryl
73-0012
2,4-D
73-0012
DDT
73-0401
Dieldrin
73-0423
EPTC
73-0134
Fenitrothion
73-0397
Mercurials
73-0647
Organochlorines
73-0012
Picloram
73-0137
Temperature
Allethrin
73-0386
Benomyl
73-0419
Chlorpyrifos
73-0440
Thiophanate
73-0419
Immunology
General
DDT
73-0113
Endrin
73-0113
Animals/experimental
Chlorobenzilate
73-0656
Dimethoate
73-0656
Fluometuron
73-0656
Methyl mercaptophos
73-0656
Monuron
73-0656
Propazine
73-0109
Pyrethrins
73-0427
Trichlorfon
73-0136
Humans
73-0079 73-0080
EPN
73-0078
Estox
73-0078
Methomyl
73-0078
MNFA
73-0078
Nabam
73-0078
Paraquat
73-0078
Propazine
73-0352
Simazine
73-0352
TDE
73-0651
2,4,5-Trichlorophenol
73-0349
Integument
Hair/fur
Human
Hexachlorobenzene
73-0632
Thallium
73-0612 73-0628
Skin
Animals/Experimental
Pyrethrins
73-0427
Human
73-0079 73-0080
BHC
73-0083
Carbamates
73-0084
DDT
73-0083
EPN
73-0078
Estox
73-0078
Hexachlorobenzene
73-0632
Methomyl
73-0078
MNFA
73-0078
Nabam
73-0078
Organophosphates
73-0076 73-0084
Paraquat
73-0078
Propazine
73-0352
Pyrethrins
73-0334
Simazine
73-0352

2,4,5-
Trichlorophenol
73-0349
Ziram
73-0070
Teeth
Animals/Experimental
Lindane
73-0700
Laws and Regulations
General
73-0281
Carbamates
73-0020
Organochlorines
73-0020
Organophosphates
73-0020
Canada
73-0266
France
Herbicides
73-0497
Germany (BRD)
73-0503 73-0506
73-0507 73-0538
DDT
73-0511 73-0516
Herbicides
73-0539
Maleic anhydride
73-0520
Mercurials
73-0520
Organochlorines
73-0520
2,4,5-T
73-0520
International
73-0531
Japan
73-0518
United Kingdom
2,4,5-T
73-0017
USA
General
73-0262 73-0268
DDT
73-0011
Mercurials
73-0258
California
73-0261
EPA
73-0532 73-0533
DDT
73-0517
USSR
73-0273
Phosmet
73-0130
Metabolism
General
Absorption
Phenamiphos
73-0686
Biotransformation
73-0642 73-0674
Chloralose
73-0707
Organochlorines
73-0502
Excretion
Methoxychlor
73-0705
Triarimol
73-0161
Algae
Absorption
Dieldrin
73-0562
Birds
General
DDT
73-0672
DDT derived
compounds
73-0672
TDE
73-0672
Distribution/Storage
73-0095
Cat
Distribution/Storage
Granosan
73-0356
Chicken
General
Fenitrothion
73-0447
Organochlorines
73-0107
Ronnel
73-0107
Distribution/Storage
Carbaryl
73-0706
2-Chloro-1-(2,4-
dichlorophenyl)
vinyl diethyl phosph
73-0143
Kerb
73-0664
Excretion
Carbaryl
73-0196
Cow
General
Benoxaphos
73-0667
2,4-D
73-0043
Hempa
73-0173
MCPA
73-0043
Organochlorines
73-0599
Silvex
73-0043
2,4,5-T
73-0043
VCS-438
73-0144
Distribution/Storage
2,4-D
73-0157
Kerb
73-0664
Silvex
73-0157
2,4,5-T
73-0157
Excretion
Carbaryl
73-0449
Propoxur
73-0449
Crab
Absorption
Mirex
73-0454
Daphnia
General
DDT
73-0660
Methoxychlor
73-0660
Dog
General
Dimethirimol
73-0126
Distribution/Storage
Granosan
73-0356
Fish
General
DDT
73-0152 73-0190
73-0660
Methoxychlor
73-0660
Absorption
Chlorpyrifos
73-0440
Dieldrin
73-0384
Biotransformation
TFM
73-0116
Distribution/Storage
73-0168
DDT
73-0188
Dieldrin
73-0188
Guinea pig
Excretion
Bromophos
73-0710
Human
Absorption

Pralidoxime
73-0151
Biotransformation
Dieldrin
73-0457
Excretion
Granosan
73-0090
Lindane
73-0066
Pralidoxime
73-0151
Thallium
73-0612
In vitro
General
Carbaryl
73-0434
Paraoxon
73-0438
Parathion
73-0362
Biotransformation
Azinphosmethyl
73-0415 73-0418
Benomyl
73-0419
Bioxone
73-0410
Carbamates
73-0163
Chlordimeform
73-0649
3-(4-Chlorophenyl)-
1-methylurea
73-0162
2,4-D
73-0166
Diazinon
73-0411 73-0413
Diazinon derived
compounds
73-0412 73-0413
Fenitrothion
73-0414
Lethane
73-0414
Mercurials
73-0695 73-0697
2-
(Methoxy(methylthio)
phosphinylimino)-3-e
73-0417
Parathion
73-0092 73-0193
73-0435
Parathion derived
compounds
73-0193
Piperonyl butoxide
73-0414
TFM
73-0116
Thanite
73-0414
Thiophanate
73-0419
Insects
Absorption
Abate
73-0420
Biotransformation
Azinphosmethyl
73-0415
Microorganisms
Atrazine
73-0547
Metham
73-0547
Prometryne
73-0547
Simazine
73-0547
Thiram
73-0547
General
73-0006
Benomyl
73-0183
Carbaryl derived
compounds
73-0111
Dalapon
73-0002
Biotransformation
Blasticidin-S
73-0283
Carbaryl
73-0408
DDT
73-0035
Dioxins
73-0293
Karsil
73-0646
Malathion
73-0641
Naptalam
73-0159
Mink
General
DDT
73-0363
Dieldrin
73-0363
TDE
73-0363
Molluscs
Absorption
Dieldrin
73-0673
Excretion
Dieldrin
73-0673
Monkey
Distribution/Storage
Fenitrothion
73-0395
Mouse
Biotransformation
Carbaryl
73-0665
DDE
73-0194
DDT
73-0194 73-0368
Dichlorvos
73-0459
Dinobuton
73-0665
Dinoseb
73-0665
Paraoxon
73-0194
Parathion
73-0194
Distribution/Storage
DDT
73-0437
Excretion
Dichlorvos
73-0459
Plants
General
Benomyl
73-0165
Absorption
Benomyl
73-0419
Dieldrin
73-0384
Thiabendazole
73-0164
Thiophanate
73-0419
Biotransformation
Atrazine
73-0146 73-0314
Bioxone
73-0410
Blasticidin-S
73-0283
Chlordane
73-0644
3-(4-Chlorophenyl)-
1-methylurea
73-0162
Cyanazine
73-0314 73-0590
Cyprazine
73-0146
2,4-D
73-0166
Diazinon
73-0663
Disulfoton
73-0680
GS-13529
73-0146
Mercurials
73-0695 73-0696
2-
(Methoxy(methylthio)
phosphinylimino)-3-e
73-0417

Propazine
73-0146
Simazine
73-0146 73-0314
Triphenyltin
73-0676
Distribution/Storage
Thiabendazole
73-0164
Excretion
Vamidothion
73-0101
Rabbit
General
Amidophos
73-0493
Biotransformation
Parathion
73-0435
Rat
General
Dimethirimol
73-0126
Parathion
73-0362
Ronnel
73-0182
Ziram
73-0131
Absorption
Carbaryl
73-0367
Biotransformation
Allethrin
73-0428
Carbaryl
73-0142 73-0421
73-0665
Carbofuran
73-0421
Chlordane
73-0421
S-2-Cyano ethyl-N-((methylcarbamoyl) oxy)thioacetimidate
73-0456
Dichlorvos
73-0458 73-0459
Dinobuton
73-0665
Dinoseb
73-0665
Mirex
73-0666
Pyrethrins
73-0428
Distribution/Storage
BHC isomers
73-0682 73-0691
73-0698
Chlordane
73-0160
DDT
73-0160
Dieldrin
73-0160
Fenitrothion
73-0114 73-0395
Lindane
73-0160 73-0682
73-0694 73-0698
Methoxychlor
73-0160
Toxaphene
73-0160
Excretion
Carbaryl
73-0421
Carbofuran
73-0421
Chlordane
73-0421
S-2-Cyano ethyl-N-((methylcarbamoyl) oxy)thioacetimidate
73-0456
Dichlorvos
73-0458 73-0459
Rodents
General
Aldrin
73-0059
Diazinon
73-0059
Endrin
73-0059
Heptachlor
73-0059
Methyl parathion
73-0059
Parathion
73-0059
Sheep
General
Benoxaphos
73-0667
Hempa
73-0173
Distribution/Storage
2,4-D
73-0157
Silvex
73-0157
2,4,5-T
73-0157
Silkworm
Biotransformation
Carbaryl
73-0409
Snail
General
DDT
73-0660
Methoxychlor
73-0660
Morbidity and Mortality Statistics
Germany (BRD)
Paraquat
73-0617
Japan
73-0077 73-0080
73-0082
Carbamates
73-0084
EPN
73-0078
Estox
73-0078
Methomyl
73-0078
MNFA
73-0078 73-0348
Nabam
73-0078
Organophosphates
73-0084 73-0358
Paraquat
73-0078
Poland
73-0001
USA
DDT
73-0332
Endosulfan
73-0332
Endrin
73-0332
Monocrotophos
73-0332
Toxaphene
73-0332
Musculoskeletal System
Diaphragm
In vitro
2,4-D
73-0687 73-0688
Muscle, striated
Human
Organochlorines
73-0609
Organophosphates
73-0609
Skeleton/bone
Animals/Experimental
DAEP
73-0404
Endrin
73-0406
Methyl mercaptophos
73-0132
Zineb
73-0653
Mutagenesis/Teratogenesis
General
73-0264
Animals/experimental
73-0701
BHC isomers
73-0691
Captan
73-0439

Carbaryl
73-0129
DFP
73-0650
Endrin
73-0406
MCPA derived compounds
73-0702
Parathion
73-0405
Thiophanate
73-0148
Thiophanate-methyl
73-0148
Zineb
73-0653
Humans
2,4,5-T
73-0085
In vitro
2,4,5-T
73-0119
Thio-TEPA
73-0372
Ziram
73-0128
Microorganisms
Dioxins
73-0094
Nervous System
General
Animals/Experimental
Abate
73-0187
Carbaryl
73-0196
Cartap
73-0147
DAEP
73-0403
DDT
73-0371
DFP
73-0167 73-0422
DNOC
73-0692
Endrin
73-0671
Fluoroacetamide
73-0402
Granosan
73-0356 73-0657
Lindane
73-0371
Metaldehyde
73-0174 73-0354
Paraoxon
73-0422
Paraquat
73-0071
Sarin
73-0167
Soman
73-0167
Human
Dichlorvos
73-0081
EPN
73-0078
Estox
73-0078
Granosan
73-0067 73-0068
73-0090
Methomyl
73-0078
MNFA
73-0078 73-0348
Nabam
73-0078
Paraquat
73-0078 73-0081
73-0347
Thallium
73-0628
Thiram
73-0068
In vitro
Allethrin
73-0386
Behavior
Animals/Experimental
6-BAP
73-0185
DDT
73-0383
DFP
73-0689
Maneb
73-0366
Thiophanate
73-0407
Human
Lindane
73-0066
Brain
Animals/Domestic
Chloralose
73-0707
Animals/Experimental
Chloralose
73-0707
Chlordane
73-0655
DDT
73-0655
DFP
73-0426
Dieldrin
73-0171
O-Ethyl-S-(2-dimethyl-aminoethyl) methyl-thiophosphona
73-0102
Sarin
73-0369
Human
2-Butyl-2-ethyl-1,3-propanediol
73-0613
Parathion
73-0637
EEG
Animals/Experimental
Fenitrothion
73-0398
Malathion
73-0398
Peripheral nerves
Human
Organochlorines
73-0609
Organophosphates
73-0609
Polyneuritis
Human
Parathion
73-0074
Thallium
73-0612
Trichlorfon
73-0074
Spinal cord
Animals/Domestic
Chloralose
73-0707
Animals/Experimental
Chloralose
73-0707
Chlordane
73-0655
Crufomate
73-0112
DDT
73-0655
Trichlorfon
73-0112
Human
2-Butyl-2-ethyl-1,3-propanediol
73-0613
New Journals
Ambio
72-0003
Photodecomposition
73-0556
Blasticidin-S
73-0283
Dieldrin
73-0558 73-0728
Endosulfan
73-0041
Fenuron
73-0042
Iodofenphos
73-0300
Monuron
73-0042
Propanil
72-0040
Tributyltin
73-0572
Triphenyltin
73-0572

Prevention
General
73-0260 73-0262
73-0275 73-0340
73-0528
Dichlorvos
73-0091
Organophosphates
73-0333
Polychlorinated biphenyls
73-0087
2,4,5-T
73-0085
Protective equipment
73-0607
Ziram
73-0070

Reproduction/Growth
General
Animals/Experimental
2-Chloro-1-(2,4-dichlorophenyl) vinyl diethyl phosph
73-0143
DDT
73-0359
Methyl mercaptophos
73-0132
Polychlorinated biphenyls
73-0359
Microorganisms
73-0098
Carbaryl
73-0443
Chlorpyrifos
73-0443
DDT
73-0177 73-0443
Lindane
73-0177
Methoxychlor
73-0177
Wildlife
DDE
73-0339
DDT
73-0339
Lindane
73-0339
Malathion
73-0339
Androgens
Animals/Experimental
DDT
73-0437
Eggshell effects
Animals/Experimental
2,4-D
73-0105
Lindane
73-0391
Wildlife
DDE
73-0346
DDT
73-0332
Endosulfan
73-0332
Endrin
73-0332
Monocrotophos
73-0332
Toxaphene
73-0332
Embryo/fetus
Animals/Experimental
DAEP
73-0404
DDT
73-0377
Endrin
73-0406
Fluoroacetamide
73-0357
Lindane
73-0377
Parathion
73-0405
Human
2,4,5-T
73-0017 73-0085
In vitro
2,4,5-T
73-0119
Fertility/sterility
Animals/Experimental
Captan
73-0439
2,4-DB
73-0355
DDT
73-0192
Lindane
73-0391
Wildlife
DDT
73-0353
Growth
Animals/Experimental
Aziprotryne
73-0652
BHC isomers
73-0682
DAEP
73-0404
Lindane
73-0682 73-0694
Mirex
73-0454
Human
Organophosphates
73-0618
In vitro
2,4-DB
73-0093
Dinoseb
73-0093
DNOC
73-0093
Microorganisms
73-0643
Benomyl
73-0640
Captan
73-0647
Carbamates
73-0647
Chlorpyrifos
73-0121
2,4-D
73-0139
Dalapon
73-0139
2,4-DB
73-0093
DDT
73-0669 73-0690
Dichlorvos
73-0669
Dinoseb
73-0093
Diuron
73-0139
DNOC
73-0093 73-0135
Fluometuron
73-0139
Granosan
73-0139
Linuron
73-0139
Mercurials
73-0647 73-0690
Nabam
73-0139
Propazine
73-0139
Hormones
Animals/Experimental
DDT
73-0117
Neonate
Animals/Experimental
Fluoroacetamide
73-0357
Placental transfer
Animals/Experimental
BHC isomers
73-0691
Human
BHC
73-0048
DDT
73-0048
Dieldrin
73-0048
Heptachlor epoxide
73-0048
Lindane
73-0048

Reproductive organs, female
Animals/Experimental
DDT
73-0693
Reproductive organs, male
Animals/Experimental
BHC
73-0446
2,4-DB
73-0355
DDT
73-0446
Dieldrin
73-0703
TDE
73-0108
Trichlorfon
73-0446
Senescence
Plant
Benomyl
73-0165
Residue Degradation
General
73-0548 73-0556
Carbaryl
73-0665
DDT
73-0544
2,2-Dimethyl-2,3-dihydrobenzofuranyl-7-N-(dimethoxy-phosp
73-0326
Dinitramine
73-0724
Dinobuton
73-0665
Dinoseb
73-0665
Methoxychlor
73-0736
Propazine
73-0323
Food and Feed
Atrazine
73-0314
BHC isomers
73-0577
Bladex
73-0314
DDT
73-0545
Lindane
73-0577
Malathion
73-0051
Monocrotophos
73-0589
Organochlorines
73-0570
Simazine
73-0314
Soil
Alachlor
73-0543
Aldrin
73-0559
Atrazine
73-0287 73-0314
73-0543 73-0547
Bladex
73-0314
Cyanazine
73-0590
Cyprazine
73-0543
Diazinon
73-0663
Dicloran
73-0325
Dioxins
73-0293
Disulfoton
73-0680
Diuron
73-0287
Fensulfothion
73-0723
Herbicides
73-0543
Malathion
73-0318 73-0563
Metham
73-0547
Organochlorines
73-0570
PCNB
73-0325
PCP
73-0284
Phosphine
73-0566
Prometryne
73-0287 73-0547
SD-15418
73-0543
Simazine
73-0314 73-0547
Sodium chlorate
73-0175
Thiram
73-0547
Trifluralin
73-0287
Zinc phosphide
73-0566
Water
Dibromochloropropane
73-0069
Herbicides
73-0709
Malathion
73-0585
Organochlorines
73-0585
Salicylanilide I
73-0600
Wildlife
Sodium chlorate
73-0175
Residue Dynamics
73-0550
Carbaryl
73-0328
DDT
73-0304
Fenitrothion
73-0045
Herbicides
73-0033
Organochlorines
73-0024
Organophosphates
73-0024
Residue Removal
General
73-0004
Food and Feed
BHC
73-0591
Blasticidin-S
73-0283
Malathion
73-0051
Organochlorines
73-0502 73-0549
Organophosphates
73-0549
Plants
Methyl bromide
73-0298
Soil
Aldicarb
73-0290
Water
Aldicarb
73-0290
2,4-D
73-0296
DDT
73-0294 73-0296
Herbicides
73-0709
Lindane
73-0296
Malathion
73-0296
Parathion
73-0296
Residues/Air
General
73-0046
Dichlorvos
73-0021
Granosan
72-0039
Lindane
73-0021
Exterior
Remote
DDE
73-0289

DDT
73-0289 73-0593
Urban
PCP
73-0313
Interior
General
Bromophos
73-0710
Trichlorfon
73-0712
Industrial
Granosan
73-0068
Maneb
73-0341
Pebulate
73-0376
Thiram
73-0068
Zineb
73-0374
Ziram
73-0070
Residues/Domestic Animals
General
Organochlorines
73-0564
Cow
DDT
73-0297
Dog
Organochlorines
73-0307
Residues/Food and Feed
General
73-0046
BHC
73-0594
Carbamates
73-0020
DDE
73-0594
DDT
73-0594
Dieldrin
73-0594
Herbicides
73-0037
Organochlorines
73-0020 73-0564
Organophosphates
73-0020
Piperonyl butoxide
73-0315
Pyrethrins
73-0315
TDE
73-0594
Triphenyltin
73-0030
Animal feed
Carbaryl
73-0328
DDT
73-0603
DSMA
73-0319
Lindane
73-0305
MAMA
73-0319
Methyl parathion
73-0603
MSMA
73-0319
Phosalone
73-0295
Polychlorinated biphenyls
73-0087
Prothoate
73-0603
Trichlorfon
73-0603
Cereals
BHC isomers
73-0577
Blasticidin-S
73-0283
Heptachlor
73-0303
Lindane
73-0577
TCA
73-0557
Dairy products
BHC
73-0316 73-0575
2,4-D
73-0043
MCPA
73-0043
Organochlorines
73-0062 73-0502
73-0551 73-0553
73-0598 73-0599
Silvex
73-0043
2,4,5-T
73-0043
Fruits
Aminozide
73-0050
DDT
73-0304 73-0545
Griseofulvin
73-0330
Methyl parathion
73-0555
Phosalone
73-0295
Thiabendazole
73-0568
Thiometon
73-0490
Meat
2,4-D
73-0157
Silvex
73-0157
2,4,5-T
73-0157
Rice
BHC
73-0049
Total diet
73-0506
Organochlorines
73-0302
Vegetables
Aldicarb
73-0290
Aldrin
72-0027 73-0595
Carbaryl
73-0047
Dacthal
73-0047
DDT
73-0329 73-0560
Diazinon
73-0026
Dieldrin
72-0027 73-0595
Dinoseb
73-0606
Disulfoton
73-0026
Endrin
73-0595
Heptachlor
73-0303
Lindane
72-0027 73-0305
73-0560
Malathion
73-0051
Methoxychlor
73-0560
Methyl bromide
73-0561
Organochlorines
73-0047
Prometryne
73-0032
Sodium arsenite
73-0054
Thiometon
73-0490
Trifluralin
73-0047
Wine
DDT
73-0545
Residues/Humans
General
73-0025
Fenitrothion
73-0075
Granosan
73-0068
Malathion
73-0075

Mercurials
73-0052
Organochlorines
73-0310
Parathion
73-0075
Polychlorinated biphenyls
73-0016
Salithion
73-0075
Thiram
73-0068
Adipose
73-0046
Organochlorines
73-0053 73-0601
Blood
Organochlorines
73-0053 73-0597
Milk
BHC
73-0048
DDT
73-0048
Dieldrin
73-0048
Heptachlor epoxide
73-0048
Lindane
73-0048
Organochlorines
73-0308 73-0311
73-0596 73-0601
Polychlorinated biphenyls
73-0311
Organs
DDE
73-0029
DDT
73-0029
Dieldrin
73-0029
Organochlorines
73-0309

Residues/Plants
General
Fungicides
73-0546
Sodium chlorate
73-0175
Cotton
Dichlorvos
73-0299
DSMA
73-0319
MAMA
73-0319
MSMA
73-0319
Naled
73-0299
Trichlorfon
73-0299
Flax
TCA
73-0557
Forest
Methyl bromide
73-0298
Tobacco
Carbamates
73-0602 73-0721
Organochlorines
73-0602 73-0721
Organophosphates
73-0602 73-0721
Phosphine
73-0327

Residues/Soil
General
73-0291 73-0586
Aldicarb
73-0290
Aldrin
73-0595
Atrazine
73-0287
BHC
73-0049
BHC isomers
73-0577
Carbaryl
73-0044 73-0047
Carbaryl derived compounds
73-0044
Chlordane
73-0061
Dacthal
73-0047
DDT
73-0061 73-0304
Dieldrin
73-0061 73-0595
Dioxins
73-0286 73-0293
Diuron
73-0287
DSMA
73-0319
Endrin
73-0061 73-0595
Heptachlor
73-0061
Heptachlor epoxide
73-0061
Herbicides
73-0037
Lindane
73-0577
MAMA
73-0319
MSMA
73-0319
Organochlorines
73-0024 73-0047
73-0571
Organophosphates
73-0024
PCP
73-0284 73-0312
Picloram
73-0331
Prometryne
73-0287
Sodium arsenite
73-0054
Sodium chlorate
73-0175
2,4,5-T
73-0312
Toxaphene
73-0061
Trifluralin
73-0047 73-0287
Triphenyltin
73-0030
3,5-Xylyl methylcarbamate
73-0044
Adsorption
BHC isomers
73-0317
Blasticidin-S
73-0283
Diuron
73-0321
Herbicides
73-0324
Lindane
73-0317 73-0320
Parathion
73-0322 73-0567
Movement
Aldrin
73-0559
Benomyl
73-0592
Fensulfothion
73-0685
Organophosphates
73-0588
Phorim
73-0685
Volatilization
DDT
73-0034
Disulfoton
73-0680
Lindane
73-0034

Residues/Water
General
73-0005 73-0014
73-0046 73-0583
Dibromochloropropane
73-0069
Lindane
72-0036
Organochlorines
73-0022
Organophosphates
73-0022

Estuaries/marshes
DDT
73-0035
Organochlorines
73-0057 73-0633
Toxaphene
73-0056
Groundwater/rain
Aldicarb
73-0290
DDT
73-0593
Malathion
73-0585
Organochlorines
73-0585
Lakes/ponds
DDE
73-0569
DDT
73-0063 73-0569
Dieldrin
73-0569
Endrin
73-0569
Lindane
73-0063
Mercurials
73-0574
Methoxychlor
73-0063
Organochlorines
73-0582 73-0584
TDE
73-0569
Oceans/seas
DDT
73-0285
DDT derived compounds
73-0285
Mercurials
73-0285
Organochlorines
73-0510
Polychlorinated biphenyls
73-0510
Rivers/streams
73-0519
DDE
73-0576
DDT
73-0605
Organochlorines
73-0584
Wastewater
Prometryne
73-0038
Residues/Wildlife
General
Aldrin
73-0626
DDT
73-0332 73-0626
Dieldrin
73-0626
Endosulfan
73-0332
Endrin
73-0332 73-0626
Monocrotophos
73-0332
Organochlorines
73-0564
Toxaphene
73-0332
Annelids
Chlorpyrifos
73-0581
Diazinon
73-0581
Birds
DDE
73-0023 73-0288
73-0338 73-0339
73-0346 73-0569
73-0604
DDT
73-0023 73-0288
73-0338 73-0339
73-0569 73-0630
Dieldrin
73-0569 73-0604
Dioxins
73-0286
Endrin
73-0569
Lindane
73-0023 73-0288
73-0338 73-0339
Malathion
73-0339
Mercurials
73-0055 73-0578
Organochlorines
73-0565 73-0578
73-0614
Polychlorinated biphenyls
73-0023 73-0578
73-0630
TDE
73-0569
Crustacea
Chlorpyrifos
73-0581
DDE
73-0579
DDT
73-0064 73-0306
73-0579
Diazinon
73-0581
Dieldrin
73-0306
Polychlorinated biphenyls
73-0064 73-0579
Eggs
BHC
73-0552
DDE
73-0338 73-0346
DDT
73-0338 73-0552
Lindane
73-0338
Organochlorines
73-0028 73-0573
Fish
2,4-D
73-0526
DDE
73-0292 73-0478
73-0579
DDT
73-0031 73-0064
73-0285 73-0292
73-0306 73-0478
73-0554 73-0579
DDT derived compounds
73-0285
Dieldrin
73-0306
Mercurials
73-0058 73-0060
73-0285 73-0574
73-0580
Organochlorines
73-0526 73-0582
Polychlorinated biphenyls
73-0064 73-0478
73-0554 73-0579
TDE
73-0292 73-0478
Mammals
Aldrin
73-0059
DDT
73-0353
Diazinon
73-0059
Endrin
73-0059
Heptachlor
73-0059
Methyl parathion
73-0059
Organochlorines
73-0565 73-0573
Parathion
73-0059
Molluscs
Chlorpyrifos
73-0581
Diazinon
73-0581
Dieldrin
73-0587
Organochlorines
73-0633

TDE
73-0587
Plankton/algae
Dieldrin
73-0562
Respiratory System
General
Animals/Experimental
BHC
73-0446
DAEP
73-0403
DDT
73-0446
DNOC
73-0692
Metaldehyde
73-0174 73-0354
Trichlorfon
73-0446
Human
Organophosphates
73-0636
Trichlorfon
73-0351
Lung
Animals/Experimental
ANTU
73-0103
Atrazine
73-0097
Prometone
73-0097
Thiophanate
73-0407
Human
Paraquat
73-0065 73-0071
73-0072 73-0336
73-0347
Upper respiratory tract
Animals/Experimental
Propazine
73-0109
Human
Ziram
73-0070
Reviews
General
73-0009 73-0277
73-0523
2,4-D
73-0526
DDT
73-0011 73-0013
73-0269
Dichlorvos
73-0021
Lindane
73-0021
Mercurials
73-0536
Organochlorines
73-0502 73-0510
73-0526
Polychlorinated biphenyls
73-0270 73-0278
73-0510
Analysis
73-0198
Carbamates
73-0233
Organochlorines
73-0233
Organophosphates
73-0233
Toxaphene
73-0716
Monitoring and residues
73-0046 73-0519
73-0556
Carbamates
73-0020
Organochlorines
73-0020 73-0022
Organophosphates
73-0020 73-0022
Toxicology and pharmacology
73-0006 73-0168
73-0266 73-0701
Chloralose
73-0707
Dalapon
73-0002
DDT
73-0429 73-0452
DFP
73-0167
Dieldrin
73-0429
Herbicides
73-0708
Organophosphates
73-0364
Sarin
73-0167
Soman
73-0167
TDE
73-0108
Sensory System
General
Animals/Experimental
Granosan
73-0356
Human
Carbamates
73-0084
Granosan
73-0067
Organophosphates
73-0076 73-0084
Taction
Animals/Experimental
DFP
73-0426
Human
Granosan
73-0068
Thiram
73-0068
Vision
Animals/Experimental
Blasticidin-S
73-0400 73-0659
Fenitrothion
73-0390 73-0679
Fenthion
73-0390 73-0658
73-0679
Organophosphates
73-0621
Human
73-0080
BHC
73-0083
2,4-D
73-0086
DDT
73-0083
Organophosphates
73-0345 73-0618
73-0620 73-0621
73-0622 73-0623
73-0624
Ziram
73-0070
Therapeutic use
General
DDT
73-0661
TDE
73-0661
Cancer
DDT
73-0153
Parathion
73-0123
TDE
73-0155 73-0651
Triamiphos
73-0123
Gilbert's syndrome
DDT
73-0429
Dieldrin
73-0429
Jaundice
DDT
73-0429
Dieldrin
73-0429
Radioprotection
S-2-(3-Aminopropylamino) ethyl phosphorothioi
73-0150
Scabies
Chlordane
73-0432
Lindane
73-0432
Toxicity/Domestic Animals
General
73-0337

Chloralose
73-0707
Warfarin
73-0608
Cow
Organophosphates
73-0635
Toxicity/Experimental Animals
General
73-0273 73-0393
73-0507
Acetoxon
73-0375
Carbamates
73-0699
Cartap
73-0147
Chloralose
73-0707
Coumaphos
73-0375
DFP
73-0638
1-(2,6-
Dichlorobenzoyl)-3-
(3,4-dichlorophenyl)
73-0677
Dichlorvos
73-0154
O,O-Dimethyl S-
alpha-isopropoxy
carbonylbenzyl phosp
73-0141
EPTC
73-0134
Fenitrothion
73-0375
Malathion
73-0141
Metaldehyde
73-0174 73-0354
Methoxychlor
73-0705
Monitor
73-0684
Paraoxon
73-0638
Pebulate
73-0376
Phenamiphos
73-0686
Phenthoate
73-0141
Picloram
73-0137
Rodenticides
73-0699
Sodium chlorate
73-0175
Thiophanate
73-0148
Thiophanate-methyl
73-0148
Toxaphene
73-0453
Triarimol
73-0161
Trichlorfon
73-0712
Zineb
73-0374
Amphibians
DDT
73-0179
Lindane
73-0179
Methoxychlor
73-0179
Birds
DDT
73-0672
DDT derived
compounds
73-0672
Dieldrin
73-0423
Phosphamidon
73-0335
TDE
73-0672
Cat
Granosan
73-0356
Phosmet
73-0130
Chicken
Carbaryl
73-0196 73-0706
2-Chloro-1-(2,4-
dichlorophenyl)
vinyl diethyl phospl
73-0143
3-Chloro-p-toluidin
73-0191
2,4-D
73-0105
DDT
73-0662
DFP
73-0650
Dieldrin
73-0662
Herbicides
73-0445
Malathion
73-0138 73-0378
Cow
Atrazine
73-0097
Crufomate
73-0112
DDT
73-0669
Dichlorvos
73-0669
Endrin
73-0671
Granosan
73-0657
Herbicides
73-0445
MSMA
73-0361
Prometone
73-0097
Trichlorfon
73-0112
Crab
Mirex
73-0454
Dog
Chlorfenvinphos
73-0450
Dichlorvos
73-0096 73-0450
Fenitrothion
73-0390 73-0679
Fenthion
73-0390 73-0679
Granosan
73-0356
Malathion
73-0450
Organophosphates
73-0621
Ronnel
73-0433 73-0450
TDE
73-0424
Thiophanate
73-0407
Trichlorfon
73-0450
Drosophila
Carbaryl
73-0129
Duck
Aldrin
73-0626
Carbamates
73-0195
DDT
73-0626
Dieldrin
73-0626
Endrin
73-0626
Organophosphates
73-0195
Eel
PCP
73-0365
Fish
73-0168
Barban
73-0430
Celatox
73-0430
Chlorbufam
73-0430
Chlorpyrifos
73-0440

DDT
73-0152 73-0188
73-0383 73-0452
Diallate
73-0430
Dichlorvos
73-0681
Dieldrin
73-0188
Diuron
73-0711
Herbicides
73-0709
Malathion
73-0681
Organophosphates
73-0441
Propazine
73-0430
Pyrazon
73-0430
Salicylanilide I
73-0600
Squoxin
73-0425
Trichlorfon
73-0681
Frog
Mercurials
73-0460
Organochlorines
73-0460
Organophosphates
73-0460
Guinea Pig
Blasticidin-S
73-0659
Fenuron
73-0704
Fluoroacetamide
73-0125
Pyrethrins
73-0427
Monkey
Dichlorvos
73-0381
Fenitrothion
73-0397 73-0398
Malathion
73-0398
Mouse
73-0394
Aziprotryne
73-0652
6-BAP
73-0185
BHC isomers
73-0127 73-0645
Captan
73-0439
Chlordane
73-0184
DAEP
73-0403 73-0404
DDE
73-0194
2,4-D derived compounds
73-0186
DDT
73-0113 73-0194
73-0359 73-0371
DFP
73-0380
Endrin
73-0113 73-0406
Fenitrothion
73-0414
Fentin hydroxide
73-0115
Fluoroacetamide
73-0357
Lethane
73-0414
Lindane
73-0371 73-0645
2-(Methoxy(methylthio) phosphinylimino)-3-e
73-0417
Methyl bromide
73-0399
Paraoxon
73-0194 73-0380
73-0670
Parathion
73-0194
Piperonyl butoxide
73-0414
Polychlorinated biphenyls
73-0359
Sarin
73-0670
Sodium chlorate
73-0176
Sodium fluoroacetate
73-0385
Thanite
73-0414
Trichlorfon
73-0380
Tricyclohexytin hydroxide
73-0115
Partridge
DNOC
73-0692
Pheasant
DDT
73-0693
DNOC
73-0692
Rabbit
BHC
73-0396 73-0446
Blasticidin-S
73-0400
DAEP
73-0403
DDT
73-0446
Dichlorvos
73-0448
DNOC
73-0692
Fenitrothion
73-0396 73-0397
73-0398
Fluoroacetamide
73-0402
Lignasan
73-0396
Malathion
73-0398
Meobal
73-0431
Methyl bromide
73-0399
Trichlorfon
73-0156 73-0446
73-0448
Rat
Abate
73-0187
Aldrin
73-0169
Aluminum phosphide
73-0436
ANTU
73-0103
Aziprotryne
73-0652
6-BAP
73-0185
BHC isomers
73-0682
Carbaryl
73-0373
Chlordane
73-0655
DAEP
73-0403
2,4-D derived compounds
73-0186
DDT
73-0117 73-0180
73-0192 73-0377
73-0655
DFP
73-0426 73-0689
Dieldrin
73-0104 73-0169
73-0171 73-0360
Endrin
73-0406
O-Ethyl-S-(2-dimethyl-aminoethyl) methyl-thiophospho
73-0102
Fenitrothion
73-0654

Fensulfothion
73-0685
Fenthion
73-0658
Fluoroacetamide
73-0402
Heptachlor
73-0451
Herbicides
73-0373
Kitazin-P
73-0172
Lindane
73-0180 73-0377
73-0682 73-0700
Malathion
73-0668
Maneb
73-0178 73-0341
73-0366
Methoxychlor
73-0180
Methyl mercaptophos
73-0132
Organochlorines
73-0373
Parathion
73-0373 73-0405
Phoxim
73-0685
Propazine
73-0109
Propoxur
73-0181
Sodium chlorate
73-0176
TDE
73-0108
Thiram
73-0444
Toxaphene
73-0683
Trichlorfon
73-0136
Zineb
73-0133 73-0178
73-0653 73-0654
Ziram
73-0133

Sheep
Atrazine
73-0097
2,4-DB
73-0355
Herbicides
73-0445
Prometone
73-0097

Snail
Copper compounds
73-0158
Fungicides
73-0158
Nitro compounds
73-0158
Trichlorfon
73-0158

Vole
BHC
73-0446
DDT
73-0446
Trichlorfon
73-0446

Toxicity/Humans
General
73-0079 73-0080
73-0343 73-0455
Carbamates
73-0084
DDT
73-0675
Dichlorvos
73-0021
Fenitrothion
73-0075
Hexachlorobenzene
73-0632
Lindane
73-0021
Malathion
73-0075
Organochlorines
73-0616
Organophosphates
73-0084 73-0342
73-0345 73-0358
73-0618 73-0619
73-0620 73-0621
73-0622 73-0623
73-0624
Paraquat
73-0065 73-0617
Parathion
73-0074 73-0075
Salithion
73-0075
TDE
73-0155
Thallium
73-0612
Trichlorfon
73-0074 73-0351

Accidental
2-Butyl-2-ethyl-1,3-propanediol
73-0613
Granosan
73-0067 73-0090
Lindane
73-0066
Organophosphates
73-0089 73-0636
Paraquat
73-0071 73-0072
73-0336 73-0347

Experimental
Dichlorvos
73-0154
Methoxychlor
73-0705
Pralidoxime
73-0151
Pyrethrins
73-0334

Intentional
Dichlorvos
73-0081
Fenthion
73-0088
Methyl parathion
73-0088
Paraquat
73-0071 73-0081
73-0336
Thallium
73-0627 73-0628

Occupational
73-0025 73-0077
73-0082
Arsenicals
73-0615
BHC
73-0083
2,4-D
73-0086
DDT
73-0083 73-0625
EPN
73-0078
Estox
73-0078
Fenitrothion
73-0344
Granosan
73-0068
Lindane
73-0625
Maneb
73-0341
Methomyl
73-0078
MNFA
73-0078 73-0348
Nabam
73-0078
Organochlorines
73-0609 73-0610
Organophosphates
73-0076 73-0333
73-0609 73-0610
Paraquat
73-0078
Parathion
73-0637
Propazine
73-0352
Simazine
73-0352
Thiram
73-0068 73-0629
2,4,5-Trichlorophenol
73-0349

Toxicity/Wildlife
General
73-0611
2,4-D
73-0526
Organochlorines
73-0526
Birds
Aldrin
73-0626
Azinphosmethyl
73-0350
2,4-D
73-0073
DDE
73-0338 73-0339
73-0346 73-0604
DDT
73-0332 73-0338
73-0339 73-0626
Dieldrin
73-0604 73-0626
Endosulfan
73-0332
Endrin
73-0332 73-0626
Lindane
73-0338 73-0339
Malathion
73-0339 73-0350
Monocrotophos
73-0332
Organochlorines
73-0614
Phosphamidon
73-0335
Toxaphene
73-0332
Fish
DDT
73-0332
Endosulfan
73-0332
Endrin
73-0332
Monocrotophos
73-0332
Toxaphene
73-0332
Mammals
DDT
73-0353
Treatment of Poisoning
General
73-0648
2-Butyl-2-ethyl-1,3-propanediol
73-0613
Chloralose
73-0707
Dichlorvos
73-0081
Granosan
73-0067 73-0090
Metaldehyde
73-0174
Organophosphates
73-0636
Paraquat
73-0065 73-0071
73-0081 73-0336
73-0617
Parathion
73-0637
Thiram
73-0629
Trichlorfon
73-0351
Warfarin
73-0608
Acetamide
Fluoroacetamide
73-0402
Atropine
Dichlorvos
73-0448
Fenthion
73-0088
Malathion
73-0378
Methyl parathion
73-0088
Organophosphates
73-0089 73-0342
73-0635
Trichlorfon
73-0156 73-0448
BAL
Methyl bromide
73-0399
Dialysis
Thallium
73-0628
Diuresis
Paraquat
73-0072
Thallium
73-0627 73-0628
Enzymes
Blasticidin-S
73-0659
Glutathione
Methyl bromide
73-0399
Fenthion
73-0658
Lignocaine
DFP
73-0422
Paraoxon
73-0422
Obidoxime
Sarin
73-0369
Oximes
Organophosphates
73-0634
Paraoxon
73-0670
Sarin
73-0670
Phospholythine
Dichlorvos
73-0448
Trichlorfon
73-0448
Pralidoxime
Malathion
73-0378
Organophosphates
73-0089 73-0342
73-0345 73-0635
Trichlorfon
73-0156
Pyrodifenium bromide
Organophosphates
73-0619
Serotonin
DFP
73-0380
Paraoxon
73-0380
Trichlorfon
73-0380
Steroids
Organophosphates
73-0631
TMB-4
Dichlorvos
73-0448
Trichlorfon
73-0448

AUTHOR INDEX

I. PERSONAL AUTHOR INDEX

Abdel-Halim, S.
73-0625
Abdel-Kader, E. M.
73-0246
Abdel-Kader, M. M.
73-0625
Abdel-Wahab, S. M.
73-0246
Abe, E.
73-0397
Abe, H.
73-0240
Abramson, F. P.
73-0714
Abruzzese, L.
73-0741
Acker, L.
73-0601
Adamescu, G.
73-0020
Adevshvili, L. G.
73-0494
Adkisson, P. L.
73-0257
Adler, C.
73-0535
Adler, I.
73-0664
Adley, F. E.
73-0055
Aebi, H.
73-0513
Ahmad, S.
73-0115
73-0649
Akaji, H.
73-0572
73-0719
Akazaki, K.
73-0240
Albone, E. S.
73-0035
Aleksandrovskiy, V. N.
73-0634
Alekseyev, A. N.
73-0357
Alekseyeva, T. I.
73-0090
Alexander, M.
73-0006
Aleyeva, I. V.
73-0353
Allegrini, J.
73-0443
Aly, O. A.
73-0737
Amma, K.
73-0185
Andrea, J.
73-0701
Andrushaytis, G. P.
73-0526
Annett, C. S.
73-0574
Antipchuk, Yu. P.
73-0446
Archer, T. E.
73-0041
Ardatova, A. N.
73-0356
73-0657
Arevad, K.
73-0338
Arfert, H.
73-0552
Armstrong, D. E.
73-0318
Arnold, E.
73-0701
Arutyunyan, R. M.
73-0372
Asanuma, N.
73-0312
Asanuma, S.
73-0309
73-0395
Astier-Dumas, M.
72-0027
Atsumi, K.
73-0396
Aucamp, P. J.
73-0306
Aue, W. A.
73-0485
Aulerich, R. J.
73-0363
Awad, O.
73-0100
Baba, N.
73-0464
Bache, C. H.
73-0232
Bahig, M. R. E.
73-0641
Bajgar, J.
73-0102
73-0369
Baker, A. D.
73-0200
Balbaa, S. I.
73-0246
Baluba, S. A.
73-0720
Bandal, S. K.
73-0665
Bankowska, J.
73-0178
Barton, J. R.
73-0582
Bass, S. W.
73-0194
Bass, W.
73-0701
Basson, N. C. J.
73-0423
Bayley, N. D.
73-0262
Bazzi, R.
73-0741
Bebeshko, V. G.
73-0109
Bejer-Petersen, B.
73-0339
Bekmayeva, A. D.
73-0473
Bel'skaya, G. F.
73-0487
Belisle, A. A.
73-0578
Belov, L. G.
73-0137
Benchev, I.
73-0234
Benson, W. W.
73-0052
73-0053
Bergen, W. G.
73-0171
Beskid, M.
73-0180
Best, L. B.
73-0073
Betteridge, D.
73-0200
Bevenue, A.
73-0480
Beynon, K. I.
73-0314
73-0589
73-0590
Bhargava, V.
73-0426
Bhatia, S. C.
73-0104
73-0145
73-0379
Bieniek, D.
73-0644
Biggar, J. W.
73-0317
Billon, J.
73-0019
Binder, F.
73-0514
Binns, W.
73-0097
Biros, F. J.
73-0745
73-0746
Bishop, Y.
73-0701
Bjerke, E. L.
73-0043
Black, A. L.
73-0417
Blanchfield, B. J.
73-0742
Blaszyk, P.
73-0611
Blend, M. J.
73-0703
Bluethgen, A.
73-0551
Bock, F.
73-0676
Bogin, E.
73-0124
73-0125
73-0465
Bojanowska, A.
73-0178
Boler, J. B.
73-0751
Bollag, J. M.
73-0111
Bontoyan, W. R.
73-0221
Bookhout, C. G.
73-0454
Booth, G. M.
73-0140
Bordelon, B. R.
73-0294
Borisenko, N. J.
72-0039
Borlaug, N. E.
73-0267
73-0530
Boros, S. J.
73-0613
Borrel, A.
73-0173
Boseova, M.
73-0693
Botvinova, L. E.
73-0211
Bouchberg, M. S. de
73-0443
Bourke, J. B.
73-0542
Boutibonnes, P.
72-0027
Brader, L.
73-0614
Bradshaw, J. S.
73-0582
Braham, J.
73-0637
Bratkowski, T. A.
73-0382
Bratt, H.
73-0126
Brewerton, H. V.
73-0050
Brikman, L. I.
73-0712
Briquet, M.
73-0099
Broadbent, F. E.
73-0325
Brodeur, J.
73-0362
Brodie, R. E.
73-0366
Bromberg, A. I.
73-0712
Brown, A. W. A.
73-0501

Brown, D. W.
73-0055
Brown, M. J.
73-0723
Brown, N. C.
73-0427
Brown, W. H.
73-0297
Bruce, W. N.
73-0660
Brueggemann, J.
73-0573
Brzezinski, J.
73-0461
Brzheskiy, V. V.
73-0129
Buchet, J.-P.
73-0123
Buck, W. B.
73-0337
Burgat, V.
73-0173
Burnett, R.
73-0064
Cabejszek, I.
73-0177
Cabral, J. R.
73-0197
Cain, J. D.
73-0047
Caldwell, R. L.
73-0280
Camargo, L. A. A.
73-0172
Canario, Jr., M. T.
73-0587
Carcur, G. E.
73-0089
Carlstrom, A. A.
73-0740
Carmody, M.
73-0071
Carrara, G.
73-0554
Carter, J. E.
73-0025
Carvalho, D. de
73-0204
Casanova, A.
73-0161
Casida, J. E.
73-0414
73-0428
73-0665
Casler, G. L.
73-0498
Casper, H. H.
73-0367
Castle, W. T.
73-0031
Causey, K.
73-0565
Cavagnol, J. C.
73-0214
73-0220
Cecil, L. K.
73-0544
Ceribashi, M.
73-0555
Cernochova, S.
73-0113
73-0693
Chang, H. S.
73-0186
73-0187
Chau, A. S. Y.
73-0229
73-0730
73-0731
Check, R. M.
73-0587
Cherpak, V. V.
72-0039
Chesters, G.
73-0318
Chiba, M.
73-0545
Chikazawa, K.
73-0595
Childers, W. F.
73-0660
Chippendale, T.
73-0689
Chopra, S. L.
73-0563
Christ, O. E.
73-0667
Churchill, II, F. C.
73-0231
Cioran, C.
73-0074
Clausen, J.
73-0371
Cliath, M. M.
73-0034
Clifford, N. J.
73-0066
Cockrell, B. Y.
73-0096
Coic, Y.
73-0546
Cojocaru, N.
73-0606
Collina, A.
73-0735
Collins, J. A.
73-0381
Collins, T. F. X.
73-0439
Cook, M. P.
73-0366
Coon, J. M.
73-0194
Cooper, J. R.
73-0385
Coppage, D. L.
73-0441
73-0633
Cordelier, C.
73-0161
Costrini, N. V.
73-0116
Craft, C. C.
73-0568
73-0722
Craig, P. P.
73-0593
Crawford, C. R.
73-0364
Cromartie, R.
73-0578
Crosby, D. G.
72-0040
73-0041
73-0484
Culbertson, J. K.
73-0583
Curry, L.
73-0585
Custot, F.
72-0027
Cutkomp, L. K.
73-0387
D'yachuk, I. A.
73-0068
D'Itri, F. M.
73-0574
Dabija, G.
73-0020
Daimatsu, E.
73-0343
Dalloz, J. P.
73-0174
Dalton, J.
73-0745
73-0746
Damodaran, V. N.
73-0379
Daniel, J. W.
73-0126
Danilenko, L. P.
73-0130
Das, D. K.
73-0247
Dauterman, W. C.
73-0415
73-0418
Davidse, L. C.
73-0419
Davidson, J. K.
73-0336
Davis, B. D.
73-0350
Davison, K. L.
73-0662
Davison, R. M.
73-0050
Davy, F. B.
73-0383
Davydova, I. F.
73-0388
De Goeij, J. J. M.
73-0569
De Pontanel, H. G.
72-0027
De Tempe, J.
73-0018
Debelyi, A. S.
73-0472
Degros, A.
73-0062
Deichmann, W. B.
73-0274
Delannoy, F.
73-0062
Delatour, P.
73-0174
Denys, A.
73-0194
Derache, R.
73-0556
Desaiah, D.
73-0387
DeVaney, T. E.
73-0533
DeWolf, E. P.
73-0578
Dickinson, J. C.
73-0361
Didier, R.
73-0119
Dimitrov, S.
73-0328
73-0603
Dishovska, Z.
73-0700
Dmitriyenko, V. D.
73-0110
Dobryanskiy, V. M.
73-0354
Doi, T.
73-0462
Doi, Y.
73-0744
Doloshitskiy, S. I.
73-0430
Domanski, J. J.
73-0752
Domsch, K. H.
73-0643
Doornbos, F.
73-0545
Dorough, H. W.
73-0666
Dorward, W. J.
73-0112
Dougherty, R. C.
73-0745
73-0746
Downey, W. K.
73-0502
Downie, J. N.
73-0391
Doyle, W. C.
73-0729
Dranovskaya, L. M.
73-0209
73-0469
Drenth, J. J.
73-0609

Drepper, F.
73-0667
Drozdova, T. A.
73-0032
Druzhinina, V. A.
73-0374
Dubranovskaya, A. A.
73-0210
Dubyagin, V. A.
73-0704
Duke, T. W.
73-0454
73-0633
Dumas, G.
73-0173
Dumitrescu, G.
73-0074
Dupuis, G.
73-0334
73-0663
Durant, C. J.
73-0056
Dutremee, C.
73-0599
Dyatlovitskaya, F. G.
73-0211
Dyck, J.
73-0338
Eberle, D. O.
73-0225
Ebing, W.
73-0548
Edgington, L. V.
73-0592
Eglinton, G.
73-0035
Egloff, D.
73-0401
Egry, T. J.
73-0728
Egyed, M. N.
73-0124
73-0125
73-0465
Ehrenberg, L.
73-0094
El-Dib, M. A.
73-0737
El-Samra, G. F.
73-0625
El-Shamy, A. M.
73-0246
El-Zawahry, Y. A.
73-0641
Eldridge, J. M.
73-0142
Elgart, M.
73-0432
Elizarova, O. N.
73-0133
Ellington, Jr., C. P.
73-0293
Elliott, M.
73-0428
Emara, A. M.
73-0625
Endo, I.
73-0405
73-0406
Endo, S.
73-0049
Ensberg, I. F. G.
73-0609
Epler, P.
73-0168
Epstein, S. S.
73-0701
Esser, H. O.
73-0663
Evans, N. C.
73-0035
Evans, R. T.
73-0214
Everaarts, J. M.
73-0614
Eyer, H.
73-0675
Ezaki, H.
73-0078
Fadow, M. P.
73-0574
Fahmy, M. A. H.
73-0326
Fakhr, I. M. I.
73-0641
Falk, H. L.
73-0389
Farah, M.
73-0249
Farmer, W. J.
73-0320
Farris, D. E.
73-0499
Fateyeva, O. F.
73-0304
Fay, R. R.
73-0057
73-0492
Fedorenko, A. P.
73-0353
73-0446
Feland, B.
73-0668
Felix, C.
73-0173
Feltz, H. R.
73-0583
Fennelly, J. J.
73-0072
Ferguson, D. E.
73-0012
Ferguson, F. F.
73-0010
Ferrando, P.
73-0106
Feung, C.-S.
73-0166
Fiala, J.
73-0113
Fielder, J. W.
73-0261
Fikes, M. H.
73-0673
Filippov, M. P.
73-0469
Fink, R. J.
73-0543
Finlayson, D. G.
73-0723
Fisher, H. F.
73-0065
Fitzgerald, M. X.
73-0072
Fitzgerald, O.
73-0072
Flickinger, F. L.
73-0626
Forbes, P. I.
73-0085
Formica, G.
73-0225
Fouda, H.
73-0484
Fouse, D. C.
73-0568
73-0732
Fouts, J. R.
73-0368
Fowle, C. D.
73-0335
Fowler, P. A.
73-0360
Foy, C. L.
73-0410
Franz, J. M.
73-0508
Frear, D. S.
73-0162
73-0163
Frei, R. W.
73-0201
73-0476
73-0477
73-0734
73-0736
Freitag, K.-D.
73-0676
Frohberg, H.
73-0617
Fuchs, A.
73-0419
Fuhriman, D. K.
73-0582
Fujimoto, C.
73-0308
73-0691
Fujiyoshi, N.
73-0394
Fukami, J.-I.
73-0411
73-0412
73-0413
Fukuchi, S.
73-0624
Fukuda, T.
73-0186
Fukuda, Y.
73-0407
Fukuto, T. R.
73-0326
73-0417
73-0420
Funakoshi, Y.
73-0159
Funazaki, Z.
73-0312
Gabica, J.
73-0052
73-0053
Gabliks, J.
73-0661
Gabr, Y.
73-0100
Gabrilevskaya, L. N.
73-0038
Gagne, J.
73-0362
Gaim, C. S.
73-0481
Gajdos, A.
73-0674
Gajduskova, V.
73-0449
Galaka, B. A.
73-0353
Galluzzi, G.
73-0741
Gamar, Y.
73-0321
Garipova, F. Sh.
73-0656
Gautam, O. P.
73-0671
Geike, F.
73-0482
Geisman, J. R.
73-0051
Gejvall, T.
73-0094
Gelder, G. A.
73-0337
Geller, I. S.
73-0656
Gensler, P.
73-0383
Gerke, H. H.
73-0505
Gheorghiu, M.
73-0074
Ghezzo, F.
73-0029
Giam, C. S.
73-0579
Giang, P.
73-0221
Gibbs, M. M.
73-0050
Gibson, J. R.
73-0666

Gielen, J. E.
73-0149
Girdhar, K. C.
73-0563
Giri, S. N.
73-0191
Gitsova, S.
73-0233
Gochfeld, M.
73-0279
Godfrain, J.-C.
73-0173
Goette, M. B.
73-0231
Gofmekler, V. A.
73-0132
Goldberg, L.
73-0271
Goldhammer, Y.
73-0637
Goldman, M.
73-0117
Gorbacheva, N. A.
73-0491
Gordiyenko, V. M.
73-0424
Goto, M.
73-0645
Goto, T.
73-0078
Goursaud, J.
73-0062
Goyer, R.
73-0670
Goza, S. W.
73-0219
Gray, L. E.
73-0164
Green, S.
73-0189
Greenblatt, R. B.
73-0632
Greger, H.
73-0606
Grigel, M.
73-0169
Grigor'yeva, N. N.
73-0453
Groff, W. A.
73-0151
Groth, W. C.
73-0222
Grushko, Ya. M.
73-0014
Grzymala, W.
73-0182
Guerra, A. A.
73-0299
Guerrant, G. O.
73-0222
73-0231
Guilbault, G. G.
73-0122
Gulat-Marnay, C.
72-0027
Gulyas, L.
73-0088
Gupta, P. K.
73-0138
Gutenmann, W. H.
73-0144
Gzhegotskiy, M. I.
73-0037
73-0430
Habib, A.
73-0625
Hackenberg, U.
73-0436
Haegele, M. A.
73-0195
Haines, L. D.
73-0664
Hall, B. V.
73-0096
Hamamoto, M.
73-0185
Hamilton, R. H.
73-0166
Hammann, I.
73-0684
Hammett, H. L.
73-0047
Hanks, A. R.
73-0579
Hannan, P. J.
73-0690
Hansen, J. C.
73-0647
Hansen, K.
73-0507
Hanyu, A.
73-0570
Harries, W. N.
73-0112
Haruta, M.
73-0308
73-0691
Hashida, K.
73-0348
73-0402
Hashimoto, Y.
73-0403
73-0404
73-0407
Hass, D. K.
73-0381
Hastings, C. R.
73-0485
Hatina, G.
73-0160
Hattori, M.
73-0624
73-0645
Hau, H. C.
72-0027
Hayashi, M.
73-0048
Hays, H.
73-0630
Hazeltine, W.
73-0346
Heaney, S. I.
73-0604
Heath, R. R.
73-0739
Hebel, R.
73-0715
Hedrick, L. W.
73-0729
Heesche, W.
73-0551
Heilbronn, E.
73-0203
Heinisch, E.
73-0716
Helwig, A.
73-0183
Hempel, D.
73-0716
Henderson, C.
73-0580
Hendrichova, E.
73-0114
Henriksson, K.
73-0154
Henry, J. L.
73-0306
Herman, J. L.
73-0043
Hermansen, P. R.
73-0339
Herrick, R. B.
73-0143
Herring, J.
73-0058
Herzel, F.
73-0584
Hetnarski, B.
73-0416
Hibberd, D. J.
73-0604
Higdon, R. S.
73-0432
Hikita, H.
73-0658
Hillers, S. A.
73-0525
Hiltbold, A. E.
73-0319
Hilton, H. W.
73-0566
Hirabayashi, M.
73-0405
73-0406
Hiro-oka, Y.
73-0395
Hoadley, E. C.
73-0456
73-0458
73-0459
Hochstrate, C.
73-0678
Hoermann, W. D.
73-0225
Hoffman, D. L.
73-0155
Hofmann, A.
73-0617
Hogan, J. W.
73-0440
Hollmann, P.
73-0505
Holmberg, B.
73-0365
Holz, D. D.
73-0440
73-0748
Homeyer, B.
73-0685
Hopcraft, J.
73-0569
Horiba, M.
73-0206
73-0207
Horiguchi, Y.
73-0398
Horwitch, P.
73-0385
Hrdina, P. D.
73-0655
Hrdina, V.
73-0369
Hudson, R. H.
73-0195
Huffaker, C. B.
73-0259
Huggenberger, F.
73-0320
Hughes, J. T.
73-0593
Hunt, E. G.
73-0332
Hunter, J. H.
73-0331
Hunter, J. M.
73-0035
Hurlbert, S. H.
73-0121
Hussain, S.
73-0094
Hutson, D. H.
73-0456
73-0458
73-0459
Hutzinger, O.
73-0475
73-0477
73-0646
73-0736
Hyndshaw, A. Y.
73-0296
Ichihashi, M.
73-0245
Ichikawa, H.
73-0347
Ide, A.
73-0284
Ide, H.
73-0347

Ide, Y.
73-0079
Ignat'yev, A. D.
73-0273
Inagaki, K.
73-0343
Inch, T. D.
73-0588
Ingls, A.
73-0580
Inkyo, K.
73-0343
Inoue, H.
73-0185
73-0239
Inoue, Y.
73-0091
Iriai, T.
73-0159
Irving, Jr., G. W.
73-0276
Ishige, T.
73-0077
Ishiguro, T.
73-0101
Ishii, K.
73-0148
Ishii, T.
73-0237
Ishikawa, T.
73-0618
73-0658
Ising, E.
73-0710
Isobe, K.
73-0695
73-0696
73-0697
Ito, K.
73-0081
Ito, N.
73-0127
Ito, R.
73-0185
73-0187
Itzuka, H.
73-0577
Ivanayevskiy, V. S.
73-0356
73-0657
Ivanchenko, V. V.
73-0213
Ivanova-Chemishanska, L.
73-0118
73-0653
Ivanova, Z. V.
73-0134
Ivie, G. W.
73-0666
Izmirova, N.
73-0118
73-0234
Jacquet, J.
72-0027
Jakl, A.
73-0369
Jamieson, W. D.
73-0475
Jamison, V. W.
73-0002
Janda, J.
73-0692
73-0693
Janes, N. F.
73-0428
Janicki, K.
73-0001
Jayaram, M.
73-0489
73-0591
Jensen, N. F.
73-0349
Jensen, S.
73-0199
73-0265
73-0365
Johannes, H.
73-0708
Johansson, D.
73-0705
Johansson, J. O.
73-0359
Johnson, A. E.
73-0097
Johnson, H. A.
73-0593
Johnson, L. F.
73-0319
Johnson, N. F.
73-0275
Johnson, R. E.
73-0500
Johnson, W. L.
73-0580
Johnston, J. M.
73-0425
Joiris, C.
73-0028
Jones, D. W.
73-0410
Joshi, D. P.
73-0523
Jukes, T. H.
73-0515
Jumar, A.
73-0717
Jung, N.
73-0552
Jurek, A.
73-0178
73-0181
73-0255
Kadis, V. W.
73-0557
Kadoum, A. M.
73-0059
Kagan, Yu. S.
73-0272
Kahn, E.
73-0258
Kaiser, T. E.
73-0221
Kallela, K.
73-0154
Kaloyanova, F.
73-0118
Kambe, Y.
73-0079
Kamel, G.
73-0625
Kamennov, N. A.
73-0466
Kamenskiy, I. V.
73-0712
Kamiji, M.
73-0238
Kamil'dzhanov, A. Kh.
73-0295
Kaminskaya, T. L.
73-0376
Kaminskis, A.
73-0151
Kanazawa, J.
73-0238
73-0239
73-0577
Kaneko, Y.
73-0431
Kani, T.
73-0308
Kapila, S.
73-0485
Kapoor, I. P.
73-0660
Kar, P. P.
73-0422
Karpenko, V. N.
73-0376
Kasai, A.
73-0309
Kashiwa, T.
73-0244
Katague, D. B.
73-0726
Kato, E.
73-0185
Kato, K.
73-0598
Kato, S.
73-0345
Kaufman, D. D.
73-0044
Kaul, R.
73-0644
Kavetskii, V. N.
73-0212
Kawaguchi, K.
73-0186
Kawahara, T.
73-0243
73-0316
Kawai, M.
73-0399
Kawakita, H.
73-0694
73-0698
Kawamura, A.
73-0390
Kawamura, H.
73-0186
73-0187
Kawano, Y.
73-0480
Kawashiro, T.
73-0695
73-0696
Kazano, H.
73-0044
Kearney, P. C.
73-0044
73-0293
Kedenburg, C. P.
73-0667
Keeney, D. R.
73-0054
Kellner, H. M.
73-0667
Kemmerling, W.
73-0708
Kemp, N. R.
73-0200
Keogh, B.
73-0071
Keymer, T. F.
73-0604
Khalifa, S.
73-0217
Khan, M. A.
73-0112
Khasanov, Yu. U.
73-0295
Khitsenko, I. I.
73-0131
Khudoley, V. V.
73-0452
Khuryev, B. B.
73-0132
Kihlstroem, J E.
73-0359
Kilbourn, E. E.
73-0699
Kilgore, W. W.
73-0725
Kimmel, F. C.
73-0428
Kimmerle, G.
73-0686
King, K. A.
73-0626
Kirchhoff, J.
73-0747
Kirihata, M.
73-0464
Kitahara, H.
73-0206
73-0207
73-0462

Kleerekoper, H. 73-0383
Klein, R. 73-0421
Klein, W. 73-0559 73-0644
Kleinrok, Z. 73-0639
Kliger, L. 73-0567
Klimmer, O. R. 73-0616
Klisenko, M. A. 73-0494
Kluge, E. 73-0547
Knaak, J. B. 73-0142
Knapek, R. 73-0683
Knight, Jr., L. A. 73-0058
Knobloch, E. 73-0693
Knowles, C. O. 73-0115 73-0300 73-0649
Ko, F. 73-0242
Kobayashi, A. 73-0049 73-0207
Kobayashi, H. 73-0311
Kobayashi, M. 73-0307
Kobayashi, S. 73-0186
Koch, R. 73-0627 73-0628
Koch, R. B. 73-0387
Kodama, J. K. 73-0381
Koehler, F. 73-0541
Koelle, W. 73-0576
Koeman, J. H. 73-0569 73-0614
Kohno, M. 73-0237
Kohsiek, H. 73-0550
Kojima, K. 73-0185
Kolodziejczyk, A. 73-0683
Koloff, W. J. 73-0007
Komissarenko, V. P. 73-0424
Konat, G. 73-0371
Kondo, T. 73-0431
Konishi, Y. 73-0084
Konrad, J. G. 73-0318
Kontek, M. 73-0496
Koransky, W. 73-0392
Korolev, A. A. 73-0375
Korte, F. 73-0558
Kortus, J. 73-0114
Koshimura, S. 73-0652
Kosmatyy, Ye. S. 73-0212 73-0303 73-0471
Kossakowski, S. 73-0156
Kovaleva, Ye. S. 73-0305
Kramer, R. E. 73-0292
Krampl, V. 73-0169
Krause, R. T. 73-0227
Krishna Menon, P. 73-0302
Kruckenberg, S. M. 73-0450 73-0463
Krupa, A. 73-0629
Krylova, N. A. 73-0208
Kuan, S. S. 73-0122
Kubacka-Szmidtgal, M. 73-0560
Kubo, H. 73-0622
Kubota, H. 73-0308
Kubota, K. 73-0311
Kudel', K. A. 73-0303
Kudo, N. 73-0358
Kuleshov, N. P. 73-0372
Kumbier, E. 73-0638
Kurmanov, I. A. 73-0448
Kurosawa, K. 73-0395 73-0397
Kuznetsova, L. I. 73-0109
Laanio, T. L. 73-0663
Lacassagne, A. 73-0108
Lakshminarayana, V. 73-0302
Lamont, T. G. 73-0578
Lamoureux, G. L. 73-0146
Lanouette, M. 73-0229
Larini, L. 73-0172 73-0204 73-0250
Larsson, A. 73-0365
Laseter, J. L. 73-0294
Laskina, V. P. 73-0038
Lauwerys, R. 73-0123
Law, S. E. 73-0607
Lawrence, J. F. 73-0201 73-0734
Lech, J. J. 73-0116
Lee, T. P. 72-0036
Lee, Y. W. 73-0060
Lees, P. 73-0707
Leesch, J. G. 73-0420
Legator, M. S. 73-0189
Leib, E. 73-0521
Leibovich, D. L. 73-0377
Leitch, R. E. 73-0486
Lellinger, D. B. 73-0021
Leng, J.-J. 73-0632
Lenon, H. 73-0585
Leroy, M. 73-0062
Letey, J. 73-0320
Levy, J. 73-0184
Lewander, K. 73-0365
Ley, R. V. 73-0588
Liebmann, H. 73-0638
Liebmann, R. 73-0716
Likovsky, Z. 73-0693
Ling, G. M. 73-0655
Lisk, D. J. 73-0144 73-0542
Liska, D. 73-0046
Liu, S. Y. 73-0111
Lloyd, J. W. 73-0437
Locke, L. N. 73-0578
Locke, R. K. 73-0216
Loefroth, G. 73-0094
Loeser, E. 73-0686
Loew, H. 73-0628
Loftus, G. 73-0542
Lohs, K. 73-0455 73-0642
Lombardo, P. 73-0728
Longchamp, R. 73-0497
Looker, J. B. 73-0221
Lorgue, G. 73-0173 73-0174
Loveridge, E. L. 73-0582
Lowe, J. I. 73-0454
Lozes, R. L. 73-0122
Lucier, G. W. 73-0421
Luczak, J. 73-0063
Luechtrath, H. 73-0615
Lundberg, C. 73-0359
Luppi Mosca, A. M. 73-0093
Luquet, F. 73-0062

Lutz-Ostertag, Y. 73-0119
Luzhichenko, N. N. 73-0704
Luzhnikov, Ye. A. 73-0634
Lyalikov, Yu. S. 73-0209 73-0469
Macek, K. J. 73-0440
MacNeil, J. D. 73-0477 73-0736
MacPherson, P. 73-0336
Maebashi, H. 73-0390
Maeda, H. 73-0702
Maehashi, H. 73-0393 73-0394 73-0431
Maier-Bode, H. 73-0506
Maillet, M. 73-0173
Maini, P. 73-0735
Majumder, S. K. 73-0489
Makarova, S. V. 73-0470 73-0487
Maki, S. 73-0175 73-0176 73-0307
Makino, T. 73-0343
Makita, T. 73-0404 73-0407
Makovskaya, Ye. I. 73-0453
Mal'tsev, P. V. 73-0067
Malec, D. 73-0639
Maleszewska, J. 73-0177
Malina, M. 73-0224
Mallet, V. 73-0476
Malone, J. D. G. 73-0071
Marciniak, C. 73-0062
Marga, K. 73-0555
Marganian, V. M. 73-0581
Markina, V. V. 73-0466
Marking, L. L. 73-0600
Martens, P. 73-0005 73-0028
Martson', L. V. 73-0070
Martynov, A. A. 73-0351
Martynyuk, N. V. 73-0430
Marugami, M. 73-0127
Maruyama, M. 73-0406
Maruyama, S. 73-0307
Massaux, F. 73-0030
Massing, H. 73-0539
Masterton, W. L. 72-0036
Masuda, T. 73-0239
Masumoto, H. T. 73-0230
Mathew, T. V. 73-0247 73-0248
Matin, M. A. 73-0422
Matokhnyuk, L. A. 73-0341
Matsu-ura, S. 73-0187
Matsumara, F. 73-0382
Matsunaga, A. K. 73-0205
Matsunaga, T. 73-0081
Matsushima, S. 73-0344 73-0397
Mattheis, I. 73-0552
Matthew, H. 73-0065
Matthies, H. 73-0638
Mattox, V. R. 73-0155
Maugh, II, T. H. 73-0534
Mawdesley-Thomas, L.E. 73-0433
Mawhinney, M. G. 73-0437
Mayer, J. 73-0114
Mayer, Jr., F. L. 73-0188 73-0748
Mazur, H. 73-0256
Mazzocchi, P. H. 73-0042
McCracken, R. M. 73-0608
McDaniel. O. S. 73-0421
McGill, A. E. J. 73-0733
McIntire, C. D. 73-0562
McIntire, G. 73-0011
McIntrye, Jr., S. C. 73-0565
McParland, P. J. 73-0608
McPhillips, J. J. 73-0438
Medved', I. L. 73-0134
Mega, T. 73-0127
Megemont, C. 73-0173
Meguro, Y. 73-0310
Meinen, V. J. 73-0218
Meinhold, K. 73-0505
Mel'nikov, N. N. 73-0277
Mel'nikova, Ye. A. 73-0330
Mel'tser, F. R. 73-0495
Mellanby, K. 73-0537
Meltzer, J. 73-0677
Merenyuk, G. V. 73-0135
Metcalf, R. L. 73-0140 73-0269 73-0660
Metelitsa, V. K. 73-0493
Meyer, F. P. 73-0636
Meyrick, B. 73-0103
Mezonnet, R. 72-0027
Michel-Ber, E. 73-0184
Mikiver, M. 73-0203
Milburn, S. E. 73-0117
Milby, T. H. 73-0333
Miles, J. R. W. 73-0226
Miles, J. W. 73-0222 73-0231
Miller. P. W. 73-0043
Miller-Ben-Shaul, D. 73-0564
Miller, A. 73-0585
Miller, J. 73-0103
Mills, A. C. 73-0317
Minasyan, S. A. 73-0329
Misato, T. 73-0283
Mishima, S. 73-0623
Mishin, G. P. 73-0090
Mita, K. 73-0396
Mitchell, E. M. 73-0724
Mitchell, J. C. 73-0334
Mitra, S. N. 73-0247 73-0248
Mitsui, Y. 73-0620
Miyabe, M. 73-0575
Miyagawa, T. 73-0645
Miyahara, K. 73-0238 73-0577
Miyamoto, Y. 73-0749 73-0750
Miyazaki, T. 73-0390
Mizoguchi, M. 73-0308
Mizuno, K. 73-0343
Moeschlin, S. 73-0648
Moilanen, K. W. 72-0040
Moku, M. 73-0243
Molberg, E. S. 73-0557
Mollenhauer, P. H. 73-0503
Mollet, M.-F. 73-0553
Monks, I. H. 73-0126

Monod, J.-L.
73-0554
Moore, J. B.
73-0315
Moore, N. W.
73-0522
Moore, W.
73-0122
Mori, T.
73-0403
73-0407
Mori, Y.
73-0343
Morikawa, O.
73-0159
Morimoto, K.
73-0691
Moriyama, H.
73-0409
Moriyama, I.
73-0347
Moroz, A. M.
73-0037
Morris, R. D.
73-0266
Mostafa, I. Y.
73-0641
Motoi, E.
73-0652
Motoyama, N.
73-0415
73-0418
Moza, P.
73-0559
Mrak, E. M.
73-0263
Mukherjee, G.
73-0248
Mulder, R.
73-0677
Mulhern, B. M.
73-0578
Mull, R. L.
73-0191
Mulla, M. S.
73-0121
Mullison, W. R.
73-0033
Mumma, R. O.
73-0166
73-0217
Munoz, V. M.
73-0089
Munson, T. O.
73-0064
Murano, A.
73-0206
73-0207
73-0462
73-0713
Murasawa, K.
73-0652
Murata, K.
73-0185
Murphy, P. G.
73-0743
Mustafa, M. A.
73-0321
Nadler, J. V.
73-0385
Nagasaki, H.
73-0127
Nagase, M.
73-0242
Nagata, H.
73-0076
Nagl, H. G.
73-0558
Nakamura, S.
73-0309
73-0395
Nakamura, T.
73-0396
Nakaoka, M.
73-0598
Namba, T.
73-0342
Narahashi, T.
73-0386
Narkiewicz-Jodko, J.
73-0026
Naydenov, N.
73-0603
73-0635
Nayshteyn, S. Y.
73-0377
Nazarewica, T.
73-0181
Nazer, I. K.
73-0041
Neal, R. A.
73-0092
73-0193
73-0435
Nearpass, D. C.
73-0323
Nebert, D. W.
73-0149
Nelson, D. L.
73-0364
Nelson, L. A.
73-0752
Nemee, Z.
73-0693
Nemteanue, S.
73-0020
Nepoklonov, A. A.
73-0493
Nesemann, E.
73-0602
73-0721
Neuhold, J. M.
73-0188
Nevstruyeva, V. V.
73-0374
Newland, L. W.
73-0057
73-0492
Newsom, H. C.
73-0724
Newsome, W. H.
73-0215
Nigo, T.
73-0695
Niki, Y.
73-0284
Nikolayev, A. I.
73-0656
Nilsson, C.
73-0479
Nimmo, D. R.
73-0442
Nishi, M.
73-0343
Nishibe, T.
73-0407
Nishimoto, T.
73-0594
73-0595
Nishimura, M.
73-0390
73-0431
Nishiuchi, K.
73-0158
Noda, K.
73-0405
73-0406
Noel, P. R. B.
73-0433
Noguchi, T.
73-0403
73-0404
73-0407
Nomura, T.
73-0081
Norkowski, S.
73-0156
Norman, S. M.
73-0568
73-0732
Novikova, K. F.
73-0495
Numata, H.
73-0405
Nuzhdina, D. P.
73-0133
O'Brien, R. D.
73-0416
O'Dwyer, W. F.
73-0071
Oba, N.
73-0623
Oberdisse, E.
73-0678
Odo, K.
73-0623
Oerberg, J.
73-0359
Oertzen, J.-A. Von
73-0510
Ogata, I.
73-0013
Ogata, S.
73-0390
73-0679
Ogawa, Y.
73-0245
Ohkawa, H.
73-0414
Ohkawa, R.
73-0414
Ohno, M.
73-0464
Ohnuma, N.
73-0407
Ohsuga, S.
73-0236
Oikawa, K.
73-0077
Okey, A. B.
73-0153
Olefir, A. I.
73-0136
Olindo, P. M.
73-0569
Olive, B. M.
73-0221
Olsson, M.
73-0365
Onishi, M.
73-0749
73-0750
Oshiba, K.
73-0682
73-0694
73-0698
Oshino, E.
73-0343
Osman, M.
73-0100
Ostarhild, H.
73-0524
Ostroumov, S. A.
73-0170
Osweiler, G. D.
73-0337
Otsuji, H.
73-0312
73-0395
Otsuka, J.
73-0621
Ottoboni, A.
73-0192
Oura, H.
73-0311
Oura, T.
73-0311
Overstreet, D. H.
73-0689
Ovseyenko, G. I.
73-0272
Oya, T.
73-0241
Oyama, J.
73-0245
Oyamada, M.
73-0159

Paez, D. M. 73-0249
Palmer, J. S. 73-0445
Palmer, K. A. 73-0189
Palut, D. 73-0182
Pantaleon, J. 73-0553
Paradowski, S. 73-0496
Parasar, G, C. 73-0378
Parish, R. C. 73-0669
Parsenyuk, L. N. 73-0210
Parsons, J. K. 73-0350
Partridge, R. 73-0401
Parys, J. 73-0251
Pasarella, N. R. 73-0738
Pashyan, R. P. 73-0329
Patel, N. R. 73-0729
Patouillet, C. 73-0690
Patrashku, F. I. 73-0045
Patulski, D. 73-0585
Paul, B. S. 73-0138
Paulson, G. D. 73-0196
Pav, J. 73-0693
Pavanello, R. 73-0029
Peardon, D. L. 73-0699
Peaslee, M. H. 73-0117
Peinkowska, A. 73-0629
Pekas, J. C. 73-0434
Pellegrini, G. 73-0141
Peloz, D. D. 73-0356
Pennings, J. H. 73-0569
Peoples, S. A. 73-0191
Perlovskaya, Z. D. 73-0704
Persson, B. 73-0023
Peters, D. A. V. 73-0655
Peterson, J. L. 73-0582
Petrocelli, R. M. 73-0205
Petrov, D. 73-0706
Petrova-Vergieva, T. 73-0653
Pettigrew, R. J. 73-0105
Pfaffli, P. 73-0154
Pfeifer, I. 73-0113
Phillips, J. A. 73-0391
Phillips, J. H. 73-0285
Phillips, L. V. 73-0729
Phillips, R. E. 73-0324
Phillips, W. E. J. 73-0160
Pickering, B. A. 73-0456 73-0458
Piechocka, J. 73-0252 73-0253
Piekacz, H. 73-0256
Pilinskaya, M. A. 73-0070 73-0128
Pitblado, R. E. 73-0592
Place, M. 73-0599
Plaiasu, D. 73-0074
Planiere, J. 73-0531
Plapp, Jr., F. W. 73-0292
Poirrier, M. A. 73-0294
Pokorna-Hanusova, D. 73-0693
Polimeno, M. 73-0093
Polin, D. 73-0363
Polizu, A. 73-0606
Polonskaya, F. I. 73-0303
Poloz, D. D. 73-0447
Pomerantz, I. H. 73-0728
Ponchet, J. 73-0640
Ponomareva, L. A. 73-0656
Portig, J. 73-0392
Potsheba, T. L. 73-0712
Poulsen, E. 73-0009
Pourtallier, J. 73-0107
Pozin, Z. S. 73-0712
Prado, A. B. 73-0204 73-0250
Prati, L. 73-0029
Preston, Jr., W. H. 73-0532
Prospero, J. M. 73-0289
Prouty, R. M. 73-0578
Pujman, V. 73-0113 73-0693
Puyear, R. L. 73-0196
Quinby, G. E. 73-0278
Quraishi, M. S. 73-0290
Rabochev, V. K. 73-0355
Raith, L. 73-0651
Rajyalakshmi, B. 73-0139
Rakhmatullayev, N. N. 73-0069
Rakhmatullayeva, M. D. 73-0340
Ramade, F. 73-0536
Ramsteiner, K. 73-0225
Ranke-Rybicka, B. 73-0179
Rao, M. P. 73-0042
Rappe, C. 73-0479
Rashev, Z. 73-0654
Ravera, O. 73-0022
Raybaud, H. 73-0554
Raymond, R. L. 73-0002
Rebmann, H. 73-0549
Reichel, W. L. 73-0578
Reid, L. 73-0103
Reimold, R. J. 73-0056
Reinbold, K. A. 73-0660
Reinert, R. E. 73-0384
Renberg, L. 73-0199
Reyes, H. D. 73-0429
Reznikov, A. G. 73-0424
Rhead, M. M. 73-0035
Richardson, A. 73-0457
Richardson, R. L. 73-0579
Richburg, P. W. 73-0565
Richou-Bac, L. 73-0553
Rickett, F. E. 73-0427
Rico, A. G. 73-0173
Rider, J. R. 73-0729
Rikimaru, T. 73-0078
Ringer, R. K. 73-0363
Rippee, K. P. 73-0582
Risebrough, R. W. 73-0630
Robel, R. J. 73-0059
Roberts, D. V. 73-0609
Roberts, T. A. 73-0604
Robinson, J. 73-0457 73-0733
Robinson, R. G. 73-0282
Robison, W. H. 73-0566
Rodgers, W. H., Jr. 73-0268
Rodionov, G. A. 73-0109
Rogatko, I. V. 73-0446
Rogers, W. M. 73-0228
Rom, R. C. 73-0504
Romina, L. I. 73-0488
Rose, F. L. 73-0562

Rosenkranz, H. S.
73-0370
Rosenkranz, S.
73-0370
Roth, H.
73-0298
Roubal, Z.
73-0693
Rousseau, M.
73-0107
Roux, M.
73-0062
Rozek, A.
73-0224
Rozgon, M. I.
73-0656
Rozin, D. G.
73-0473
Ruedel, R.
73-0687
73-0688
Ruf, H.
73-0576
Rump, S.
73-0167
Rusiecki, W.
73-0461
Russell, R. W.
73-0689
Ryazanova, R. A.
73-0374
Saad, W. A.
73-0172
Sackett, W. M.
73-0579
Sadykov, R. E.
73-0355
Safe, S.
73-0475
73-0646
73-0736
Saha, J. G.
73-0060
Saito, M.
73-0077
73-0622
Saito, T.
73-0101
Saive, R.
73-0098
Sakabe, Y.
73-0575
Sakagami, Y.
73-0572
73-0719
Sakaguchi, H.
73-0681
Sakai, M.
73-0147
Sakamoto, F.
73-0284
Sakharov, Yu. I.
73-0015
Saltzman, S.
73-0322
73-0567
Salvador, R. L.
73-0670
Sand, P. F.
73-0061
73-0586
Santi, R.
73-0141
Sapundzhieva, L.
73-0603
Sare, W. M.
73-0085
73-0086
Sarova-Pinhas, I.
73-0637
Sasaki, K.
73-0397
Sasaki, S.
73-0310
Sasaki, T.
73-0187
Sasinovich, L. M.
73-0272
Sasse, K.
73-0540
Sata, M.
73-0413
Saucier, M.
73-0670
Savage, E. P.
73-0016
Schiller, M.
73-0628
Schindler, R. L.
73-0463
Schinzel, A.
73-0519
Schmidt, P.
73-0113
Schoebel, A.
73-0709
Schroeder, R. S.
73-0729
Schulte, E.
73-0601
Schulz, D.
73-0711
Schuphan, I.
73-0548
Schwemmer, B.
73-0224
Scott, H.
73-0324
Scuka, M.
73-0386
Seba, D. B.
73-0289
Sedokur, L. K.
73-0444
Seehofer, F.
73-0602
73-0721
Seiber, J. N.
73-0484
Sekhon, K. S.
73-0523
Sell, J. L.
73-0662
Semencheva, E. M.
73-0109
Senda, I.
73-0311
Senges, J.
73-0687
73-0688
Serum, J. W.
73-0144
Shabliy, V. Ya.
73-0273
Shamray, P. F.
73-0453
Sharma, K. P.
73-0523
Sharma, R. D.
73-0671
Sharma, S. C.
73-0104
73-0379
Sheets, T. J.
73-0291
73-0752
Sherman, M.
73-0143
Shevchenko, L. S.
73-0353
Shibata, E.
73-0185
Shigan, S. A.
73-0375
Shigematsu, H.
73-0409
Shimabukuro, R. H.
73-0146
Shimada, J.
73-0152
Shimanko, I. I.
73-0634
Shimano, N.
73-0749
73-0750
Shimazaki, K.
73-0397
Shinoda, H.
73-0081
Shinozaki, C.
73-0241
Shishido, T.
73-0411
73-0412
73-0413
Shlosberg, A.
73-0125
Shrivastava, S. P
73-0300
73-0417
Shroyt, I. G.
73-0110
Shtenberg, A. I.
73-0373
Shtyler, S. Yu.
73-0468
Shubert, J.
73-0264
Sidell, F. R.
73-0151
Siegel, M. R.
73-0165
Silva, H. C.
73-0204
Simon, D.
73-0670
Simon, J.
73-0096
Sinclair, J. B.
73-0164
Singhal, R. L.
73-0655
Skoblin, A. P.
73-0032
Skoblina, L. P.
73-0032
Smead, C. F.
73-0214
Smith, A. E.
73-0557
Smith, C. G.
73-0437
Smith, G. R.
73-0604
Smith, J. T.
73-0668
Smith, M. T.
73-0437
Soboleva, D. A.
73-0487
Sobotka, T. J.
73-0366
Soderquist, C. J.
73-0484
Soedergren, A.
73-0095
73-0288
Somora, J.
73-0113
Sosnierz, M.
73-0683
Soulie, B.
73-0443
Spencer, W. F.
73-0034
Spengler, D.
73-0717
Sprott, J. M.
73-0499
Srivastava, D. N.
73-0378
St. John, Jr., L. E.
73-0232
Staerk, H.
73-0561
Stafford, L. E.
73-0146
Stahr, H. M.
73-0337

Staib, A. H.
73-0636
Stalling, C. D.
73-0059
Stander, G. H.
73-0306
Stanislawska, J.
73-0177
Stanley, C. W.
73-0726
73-0727
Starr, Jr., H. G.
73-0066
Steevens, D. R.
73-0054
Stein, M.
73-0733
Steller, W. A.
73-0738
Stephan, F.
73-0528
Stephenson, M. F.
73-0574
Stevens, J. T.
73-0438
Stevenson, A.
73-0223
Stickel, L. F.
73-0672
Stieglitz, L.
73-0576
Stiles, W. B.
73-0672
Stitzel, R. E.
73-0438
Stobbe, E. H.
73-0331
Stoimenov, V.
73-0706
Stoyanov, Kh.
73-0451
73-0635
Stoydin, G.
73-0314
73-0590
Street, J. C.
73-0188
Strokov, Yu. N.
73-0355
Stull, J. W.
73-0297
Suess, A.
73-0561
Sugaya, A.
73-0082
73-0083
73-0310
Sugimoto, T.
73-0400
73-0659
Sugiyama, F.
73-0409
Sullivan, L. J.
73-0142
Sumner, A. K.
73-0060
Supin, G. S.
73-0213
73-0470
Suzuki, Y.
73-0310
Swanson, H. R.
73-0162
73-0163
Sy, M.
73-0173
Syrowatka, T.
73-0178
73-0181
73-0255
Szabo, S.
73-0631
Szczurek, Z.
73-0683
Szutowski, M.
73-0461
Szyszkowska, A.
73-0180
Tai, H.
73-0061
73-0586
Takada, T.
73-0081
Takagi, K.
73-0283
Takahashi, J.
73-0570
Takahashi, N.
73-0343
Takaku, I.
73-0619
Takamatsu, M.
73-0078
Takano, J.
73-0241
Takano, T.
73-0464
Takano, Y.
73-0080
73-0396
Takanuma, S.
73-0571
Takase, I.
73-0236
73-0680
Takeba, K.
73-0308
73-0691
Takeda, M.
73-0695
73-0696
73-0697
Takeshige, M.
73-0079
Takeshita, R.
73-0719
Talanov, G. A.
73-0305
73-0448
73-0753
Talbott, T. D.
73-0214
Taliercio, Y.
73-0107
Tallant, M. J.
73-0142
Tamagawa, S.
73-0159
Tamarov, B.
73-0603
Tamura, O.
73-0620
Tan, K.
73-0159
Tanabe, H.
73-0695
73-0696
Tanabe, T.
73-0652
Tanaka, F. S.
73-0162
73-0163
Tanaka, H.
73-0431
Tanihata, T.
73-0187
Tarka, Z.
73-0629
Tarkov, M. I.
73-0135
Tatsumi, M.
73-0310
Taue, S.
73-0594
73-0595
Tendille, C.
73-0546
Terracini, B.
73-0197
Terry, K.
73-0730
Terui, Y.
73-0396
Tessari, J.
73-0016
Testa, M. C.
73-0197
Thielemann, H.
73-0722
Thier, H.-P.
73-0718
Thomas, J. A.
73-0437
Thompson, M.
73-0200
Thompson, P. H.
73-0608
Thornton, J. S.
73-0726
73-0727
Thorsell, W.
73-0203
Tilemans, E.
73-0024
Tillmann, P.
73-0627
73-0628
Timchenko, L. A.
73-0135
Tindle, R. C.
73-0748
Tjioe, P. S.
73-0569
Toida, S.
73-0186
73-0187
Tokareva, T. G.
73-0357
Tokoro, T.
73-0621
Tolle, A.
73-0551
Tomii, S.
73-0127
Torre, C.
73-0650
Toth, G.
73-0088
Towers, G. H. N.
73-0334
Toyoda, I.
73-0101
Tramier, P.
73-0640
Trei, J. E.
73-0669
Tret'yak, M.G.
73-0471
Triolo, A. J.
73-0194
Trondina, G. A.
73-0447
Troskho, F. V.
73-0472
Tsaregorodtseva, G. N.
73-0753
Tscheu, M.
73-0709
Tsubouchi, H.
73-0575
Tsuda, H.
73-0680
Tsujino, Y.
73-0490
Tsukahara, I.
73-0624
Tsukano, Y.
73-0049
Tubb, R. A.
73-0673
Tucker, R. K.
73-0195
Turkova, V. G.
73-0090
Turov, I. S.
73-0357

Tyszkiewicz, K.
73-0427
Uchida, A.
73-0077
Ueda, K.
73-0390
73-0399
Uete, T.
73-0749
73-0750
Ulfstrand, S.
73-0095
73-0288
Underwood, J. G.
73-0327
Unterstenhoefer, G.
73-0529
Uporova, G. I.
73-0468
Ushio, F.
73-0308
Usui, K.
73-0411
73-0413
Utley, D.
73-0588
Uyeta, M.
73-0594
73-0595
Van Daalen, J. J.
73-0677
Van Den Berg, G. A.
73-0419
Van Kampen, K. P.
73-0097
Van Velzen, A. C.
73-0672
Vasil'yev, V. P.
73-0303
Vasilos, A. F.
73-0110
Vayntraub, F. P.
73-0470
Vekshteyn, M. S.
73-0131
Vengerskaya, Kh. Ya.
73-0474
Venkataraman, G. S.
73-0139
Venkitasubramanian,T.A.
73-0104
73-0145
73-0379
Venot, C.
73-0554
Vestweber, J. G.
73-0450
Vierkoetter, S.
73-0538
Viktora, L.
73-0113
Virtamo, M.
73-0154
Visek, W. J.
73-0703
Visweswariah, K.
73-0489
73-0591
Vitvitskava, B. R.
73-0375
Voiculescu, V.
73-0074
Volkova, A. P.
73-0712
Von Stryk, F. G.
73-0483
Voronkina, T. M.
73-0712
Voytenko, G. A.
73-0376
Vrochinskiy, K. K.
73-0130
Vulchovski, Ya.
73-0706
Vuleva, R.
73-0328
73-0603
Wada, H.
73-0598
Wada, T.
73-0571
Wagenbreth, D.
73-0547
Wagner, F.
73-0561
Wahner-Roedler, D.
73-0392
Wall, Jr., W. J.
73-0581
Wallnoefer, P. R.
73-0646
Walsh, D. F.
73-0440
Walsh, L. M.
73-0054
Wang, C. H.
73-0325
Wang, C. M.
73-0386
Ware, Jr., J. E.
73-0699
Wargo, Jr., J. P.
73-0664
Warnick, S. L.
73-0025
Watanabe, F.
73-0652
Watanabe, H.
73-0284
Watanabe, M.
73-0244
Watanabe, S.
73-0598
Watanobe, S.
73-0075
Wayne, R. S.
73-0222
Weber, J. B.
73-0291
Weed, S. B.
73-0291
Weihe, M.
73-0338
73-0339
Weisgerber, I.
73-0559
Wellinga, K.
73-0677
Wells, M. R.
73-0190
Westfahl, M. E.
73-0059
Wetters, J. H.
73-0043
White, D. A.
73-0582
White, E. R.
73-0725
Whitehead, C. C.
73-0105
73-0391
Whiting, F. M.
73-0297
Wiadrowska, B.
73-0178
Wierenga, M. E.
73-0260
Wiersma, G. B.
73-0061
73-0586
Wiessmann, B.
73-0627
73-0628
Wilbert, H.
73-0509
Willett, L. B.
73-0087
Williams, C.
73-0421
Williams, D. T.
73-0742
Williams, I. H.
73-0723
Willson, H. R.
73-0121
Wilson, A.
73-0609
Wilson, A. J.
73-0454
Winter, R.
73-0627
73-0628
Witham, F. H.
73-0166
Wojcik, J.
73-0179
73-0180
Wolcott, R. M.
73-0193
Wolfenbarger, D. A.
73-0299
Wolff, J. P.
73-0198
Wolter, D.
73-0605
Wong, M. K.
73-0481
73-0579
Woods, Jr., L. A.
73-0031
Woodwell, G. M.
73-0593
Woolson, E. A.
73-0293
Worden, A. N.
73-0433
Wotzba, J.
73-0709
Wright, A. N.
73-0314
73-0589
73-0590
Wyllie, J.
73-0053
Wysocka-Paruszewska, B.
73-0120
Yajima, T. W.
73-0205
Yakusheva, O. V.
73-0657
Yamada, K.
73-0652
Yamada, T.
73-0598
Yamagishi, T.
73-0308
73-0691
Yamaguchi, I.
73-0283
Yamamoto, I.
73-0414
Yamamoto, T.
73-0185
Yamaoka, T.
73-0490
Yamasaki, A.
73-0185
Yamashita, T.
73-0237
Yamazaki, H.
73-0397
Yamazoe, H.
73-0652
Yamura, Y.
73-0307
Yanagisawa, T.
73-0397
73-0398
Yanev, S.
73-0380
Yao, B. C.
73-0051
Yap, H. H.
73-0387
Yarbrough, J. D.
73-0190
Yarish. W.
73-0557

Yaron, B.
73-0322
73-0567
Yasaka, W. J.
73-0172
Yasuda, M.
73-0702
Yasuhara, K.
73-0308
Yelizarov, G. P.
73-0352
Yoneda, H.
73-0205
Yonemura, I.
73-0406
Yotov, N,
73-0635
Yu, C.
73-0140
Yuhas, J. M.
73-0150
Yuki, F.
73-0206
73-0207
Zabbia, Jr., A. J.
73-0165
Zadrozinska, J.
73-0252
73-0254
Zaki, A. Y.
73-0246
Zakordonets, V. A.
73-0032
Zatserkovskiy, V. A.
73-0303
Zawolkow, G. A.
73-0689
Zazhivilov, A. G.
73-0460
Zelinskaya, O. V.
73-0474
Zielhuis, R. L.
73-0610
Zitko, V.
73-0478
Zlateva, M.
73-0700
Zsiga, I.
73-0088
Zubairi, M. Y.
73-0408
Zuberi, R.
73-0408
Zuccarelli, M.
73-0443
Zwolinska-Sniatalowa, Z.
73-0026
Zycinski, D.
73-0063

Fertilizers and Agr. Chem. Sub-Council.,
Nat. Ind. Pollut. Contr. Council
73-0281
Int. Ass. on Occup. Health
73-0610
Panel on Hazardous Trace Substances
73-0270
Royal Swedish Academy of Sciences
72-0003
Study Group Min. Health Welfare for Organochlorine Pesticides
73-0596
73-0597
Yamaguchi Pref. Res. Inst. of Health
73-0313

73-0497. Longchamp, R. (Lab. de Malherbol., Inst. Nat. de Rech. Agron., Versailles, France). **Les methodes d'essai des herbicides en France en vue de leur homologation. [Herbicide test methods in France with regard to the official approval of herbicides.]** *Agron. Trop. (Paris) 27(5): 553-559; 1972.* (French)

The test procedures required in France for the official approval of herbicides are reviewed. The efficiency, active dose, sensitivity or resistance of certain species, as well as toxicity and persistence of new herbicides are investigated in comprehensive field and laboratory tests. The complete trial before possible official approval usually takes three years.

73-0498. Casler, G. L. (Cornell Univ., Ithaca, NY). **Economic and policy implications of pollution from agricultural chemicals: comment.** *Amer. J. Agr. Econ. 54(3): 534-536; 1972.* (3 references)

In a Letter-to-the-Editor commenting on a previous article (see Abstract no. 72-0904), it is stated that the acreage allotment system of food supply control has not necessarily increased the total pollution from crop production or even the pollution from other specific chemicals. Although substitution of land for chemicals would result in a reduction of pollution under some circumstances, it is doubtful that a market quota system would actually result in that substitution as proposed by Farris and Sprott.

73-0499. Farris, D. E.; Sprott, J. M. (Texas A. & M Univ., College Station, TX). **Economic and policy implications of pollution from agricultural chemicals: reply.** *Amer. J. Agr. Econ. 54(3): 536; 1972.* (5 references)

A communication regarding the necessity of a basic change in farm policy (from acreage controls to market quotas) to reduce applications of polluting chemicals is defended. The critic's conclusion (see Abstract no. 73-0498) that total pollution resulting from crop production might not be reduced by substitution of market quotas is invalid. Even though more acreage may be planted, the amount of chemicals used per acre would be reduced, concentration being an important factor in pollution. The fact that the existing program causes the producer to face the same product/chemical price ratio for the last units of higher and higher price per acre yields, in contrast to the proposed program, was overlooked.

73-0500. Johnson, R. E. (Office of Environ. Quality, Bur. of Sport Fish. and Wildl., U. S. Dept. of the Interior, Washington, DC 20240). **Insecticides and the environment.** *Amer. J. Trop. Med. Hyg. 21(5): 825-828; 1972.* (21 references)

Some of the reasons for public resistance to the use of more recent chemical insecticides were discussed at an Inter-American Malaria Research Symposium. Since these insecticides are not naturally occurring, most living organisms have not had time to adapt to them although resistance has developed among some insect species. High toxicity or persistence characterize many of them; magnification in the food chain, storage in fatty tissue and mobility of many of the compounds cause concern. It is estimated that 25% of the total DDT compounds produced have been deposited in the sea. Effects on reproduction or food supplies in a number of species, mortalities, physiological changes, and adverse economic effects have all been observed in the last 30 years. Carelessness, ignorance, and indifference have also caused trouble with chemical insecticides.

73-0501. Brown, A. W. A. (Vector Biol. and Control, World Health Organization, 1211 Geneva 27, Switzerland). **The ecological implications of insecticide usage in malaria programs.** *Amer. J. Trop. Med. Hyg. 21(5): 829-834; 1972.* (16 references)

In programs of malaria control, the development of vector resistance has brought about consideration of larvicides versus adulticides, of space-spraying methods such as ultra low volume versus residual adulticides, and of biological control in addition to chemical control to benefit the environment. DDT applied indoors in malaria programs contributes to only a minor portion of the world contamination because it is not mobile. Effects on wildlife from house spraying have not been documented. Cattle are now being sprayed because toxicity appeared to be quite low. Cats have occasionally been poisoned by such applications as have house mice and roof rats. Valuable predator insects decreased although it is desirable to eliminate all insects within the household. Acceptable substitutes for DDT or dieldrin in domestic application are rare. The most promising organophosphate, abate, and the suitable carbamates are considerably more expensive. With resistance to residual adulticides, substitution of larvicides has been attempted, but application rates must be adjusted to insure minimal damage to other organisms. Agriculture uses 80 times as much insecticide in a given area as a malaria program, and most of the pollution and development of resistance have been attributed to agricultural applications of DDT.

73-0502. Downey, W. K. (Nat. Dairy Res. Cent., Agr. Inst., Fermoy, Co. Cork, Ireland). **Pesticide residues in milk and milk products, in particular the synthetic organic insecticides.** *Ann. Bull. Int. Dairy Fed. Part II, 1972, 51 pp.*

A review is presented of the material on pesticide residues pertinent to the milk industry. Responses to the International Dairy Federation's questionnaire in 1971 indicated that levels of DDT and BHC (the major contaminants), aldrin/dieldrin, and heptachlor epoxide in dairy products in 19 countries pose no definite hazard to man. The easiest means of reducing contamination is to discontinue direct treatment of dairy cattle dwellings

with organochlorines on a world-wide basis. Substitution of biodegradable pesticides for these purposes probably would not increase the cost of milk. Although pasteurization has little effect on organochlorine residues, heat treatment, condensation, removal of cream, and other processes do significantly lower them.

73-0503. Mollenhauer, P.H. (Bundesministerium fuer Jugend, Familie und Gesundheit, Bonn-Bad Godesberg, Germany). **Toxikologische Bewertung und Verzehrserhebungen als Grundlagen fuer die Festsetzung zulaessiger Hoechstmengen von chemischen Stoffen in Lebensmitteln. [Toxicologic evaluation and food consumption questionnaires for establishing permissible maximum quantities of chemical substances in foods.]** *Arch. Lebensmittelhyg. 23(8): 169-172; 1972.* (6 references) (German)

A knowledge of the type and quantity of residues present in food and the quantities of these foods consumed on the average by various population groups is essential for the establishment of standards for chemicals in foods. This is true of pesticides, as well as other substances such as food additives, animal feed additive-derived substances, drugs, and environmental contaminants. In Germany such studies have been conducted by the Deutsche Forschungsgemeinschaft (German Research Association) and the joint FAO/WHO expert committees for food additives and pesticides. Toxicologic studies provide an "acceptable daily intake" (ADI) figure, based on the no-effect level found in animal studies. This no-effect level is divided by a safety factor, generally of 100, to compensate for some uncertainties in extrapolation from animals to man. The toxicologic studies are then supplemented by the food consumption studies. Due to the incomplete nature of many studies and in order to avoid removing large numbers of foods from the market, various categories of provisional ADI figures have been defined. The expert committee for pesticide resides has defined such terms as negligible residue, unintentional residue, practical residue limit, ADI, conditional ADI, experimentally negligible daily intake, tolerance, and provisional tolerance.

73-0504. Rom, R.C. (Peach Substation, Nashville, TN). **Herbicide effects on surface soil in orchards and rainfall infiltration.** *Arkansas Farm Res. 21(4): 5; 1972.*

The effects of a pre-emergence herbicide program, one or two applications of the post-emergence herbicide, paraquat, and no weed control, on infiltrability of soil in a peach orchard were determined by measuring the electrical resistance in buried gypsum blocks. After each rainfall, less moisture was absorbed and redistributed to the 8 and 12 inch depth with the bare soil surface (pre-emergence treatment). In contrast, weedy mulch plots (paraquat-treated) were rewetted to approximately field capacity at the 12 inch depth when the rainfall exceeded 1 inch. The use of an in-the-row herbicide band of minimum width that provides adequate weed control while leaving dead weed debris on the surface to protect it from rain impact and sealing can preserve the infiltrability of orchard soil surface. The post-emergence treatment used was shown to be a satisfactory method for accomplishing this. In other studies, proper timing of pre-emergence herbicide applications gave the same results.

73-0505. Meinhold, K.; Hollmann, P.; Gerke, H.H. (Inst. fuer Betriebswirtschaft der Forschungsanstalt fuer Landwirtschaft, 33 Braunschweig, Germany). **Oekonomische Aspekte des Umweltschutzes in der pflanzlichen Produktion. [Economic aspects of environmental protection in plant production.]** *Ber. Landwirt. 50(2): 209-219; 1972.* (15 references) (German)

Farmers find it necessary to use all available means of improving the efficiency of production due to the economic pressures of competition, the price-cost differential, and rising income expectations. Such means either include or indirectly entail the use of chemical pesticides. The use of production aids such as pesticides and fertilizers may impair product quality either by influencing the quantitative ratios of natural plant components or by introducing residues of foreign substances. Other environmental effects include upsets in the soil microflora, eutrophication of surface waters, contamination of groundwater with residues and degradation products, and extermination of some plant and animal species. Various measures for reducing the quantities of production aids used have been proposed, including raising the costs of pesticides and fertilizers or placing limitations on the quantities which may be used; however, any measure taken must be thoroughly studied before implementation. The economic and environmental consequences of agricultural regulation must be examined.

73-0506. Maier-Bode, H. (Pharmakol. Inst. der Rheinischen Friedrich-Wilhelms-Universitaet Bonn, 53 Bonn, Germany). **Umfang und Art der Pestizidrueckstaende in Lebens- und Futtermitteln. [Quantities and types of pesticide residues in foods and feeds.]** *Ber. Landwirt. 50(2): 348-358; 1972.* (29 references) (German)

The West German Pesticide Tolerance Law of 1966 contains tolerance values for 92 pesticidal active ingredients in or on various foods of vegetable origin; an updated version of this law is being prepared. Many fruits and vegetables in West Germany are imported and should be monitored along with domestically grown food. Out of 2000 fruit and vegetable samples tested in Rheinland, 2.5% of the vegetables and 3.6% of the fruits contained pesticide residues in excess of the tolerance levels. Residues in prepared foods are lower since pesticides are removed by washing and cooking. Foods of animal origin contain persistent organochlorine residues

from applications of previous years. West Germany has not yet passed a law governing pesticide residue tolerances in foods of animal origin. Theoretical zero tolerances for pesticide residues have been established for baby and dietetic foods in Germany although results of actual residue analyses show that such demands are impractical. Animal feeds often contain fairly large quantities of residues because of the rapid harvesting of fodder after treatment.

73-0507. Hansen, K. (Max von Pettenkofer Inst. des Bundesgesundheitsamtes, Abt. Toxikol., Berlin-Dahlem, Germany). **Die toxikologische Beurteilung von Pestizidrueckstanden. [The toxicologic evaluation of pesticide residues.]** *Ber. Landwirt. 50(2): 383-391; 1972.* (German)

In West Germany the plant protection law states in general terms that the use of pesticides is to be approved only if it can be reasonably expected to cause no injuries. The pesticide tolerance regulations provide quantitative reference figures by establishing permissible residue levels in crops. Through these measures the legal prerequisites for health protection are provided. Animal experiments form the foundations of toxicologic evaluation. Dosages at which distinct toxic effects are observed, as well as dosages at which toxic effects are not perceptible, must be included in the studies in order to establish a no-effect level. The no-effect level, determined from animal experiments, is multiplied by a safety factor to obtain an acceptable daily intake value. This value is then compared with the average actual intake of the pesticide per day. If the actual residue level is unacceptable, attempts may be made to reduce it. If it is acceptable, the use of the treated food is permitted as long as the treatment is applied in accordance with good agricultural practice, and the residue value is entered into the tolerance regulations.

73-0508. Franz, J. M. (Inst. fuer Biol. Schaedlingsbekaempfung der Biol. Bundesanstalt fuer Land- und Forstwirtsch., 61 Darmstadt, Germany). **Biologische Schaedlingsbekaempfung. [Biological pest control.]** *Ber. Landwirt. 50(2): 417-425; 1972.* (18 references) (German)

Biological pest control may comprise either the direct propagation and field introduction of the enemies of pest species or the creation of ideal conditions for propagation of existing enemies. Biological control generally aims at reducing pest damage to a negligible level rather than eliminating it completely. Natural control mechanisms are often density-dependent so that their effectiveness increases as the pest populations rise. One exception to this rule is the use of genetically damaged, i.e., sexually sterilized members of a species to interfere with reproduction; this technique is more effective at lower population levels. Biological control has the best chance of success where the continuity of occupation by predators, parasites, and pathogens of pest species is ensured by a permanent residual population of host organisms.

73-0509. Wilbert, H. (Inst. fuer Pflanzenpathol. und Pflanzenschutz der Univ. Goettingen, Entomol. Abt., 34 Goettingen, Germany). **Integrierter Pflanzenschutz–Pest Management. [Integrated control–pest management.]** *Ber. Landwirt. 50(2): 426-440; 1972.* (128 references) (German)

The demand for integrated control grew out of the realization that one-sided pest control measures are often self-defeating, as well as uneconomical and damaging to the environment. A pest population and the factors influencing it can be viewed as a cybernetic system containing at least one control loop (the population density/birth-death loop) in which a negative feedback is produced by density-dependent control factors. This system interacts with the system of factors which determines the economic injury level by way of the control measures applied. The optimal decision concerning application of individual control measures can often be made only when a reliable predictor of crop damage is available, leaving sufficient time for application of controls. However, it is difficult to make accurate predictions concerning pest population developments due to the complexity of the systems involved. Improved data collection and processing methods developed in the past decade promise better predictability. Chemical measures will continue to form a vital part of pest management plans, and development of preparations with improved specificity is a fruitful research direction.

73-0510. Oertzen, J.-A. von (Sektion Biologie, Univ. Rostock, Rostock, DDR). **Die Meeresverschmutzung–ein Problem der Meeresbiologie. [Marine pollution–a problem of marine biology.]** *Biol. Rundsch. 10(1): 1-18; 1972.* (109 references) (German)

Current findings on marine pollution are reviewed and trends pointed out. Among the wide variety of oceanic pollutants, pesticides, especially DDT, are widely distributed in the oceans. Approximately 2.78×10^4 tons of DDT are introduced into the oceans each year via precipitation and runoff. Chlorinated hydrocarbons interfere with photosynthesis and reproduction in diatoms, important components of the phytoplankton. Marine animals which are used as human food and which serve as purifiers by filtration accumulate large quantities of organochlorines from water. Pesticides are often most harmful to the young organisms which jeopardizes the reproductive cycle. Polychlorinated biphenyls are also significant contaminants in the Baltic region. Problems to be systematically attacked by international working groups on marine pollution include development of a monitoring system, formulation of a pollution index, resolving the fate of chemicals

in the marine ecosystem, and preparing for emergency waste handling in case of disasters.

73-0511. Anonymous. **DDT-Gesetz. [The DDT law.]** *Bundesgesundheitsblatt 15(19): 281; 1972.* (German)

Principal provisions of the West German DDT law, effective Nov. 1972, are presented. A general ban on the production, import, export, sale, purchase, and use of DDT and DDT products was imposed. Permission for the exceptional use of DDT and DDT products for research, investigation and experimental purposes, the synthesis of other substances, and control of *Pediculidae, Monomorium pharaonis,* and *Cimex lectularius* (except in humans, vertebrates, stables, and areas for processing, handling, and selling foodstuffs) will be granted by the German Public Health Administration until Dec. 31, 1975.

73-0512. Anonymous. **Polychlorbiphenyle (PCB) als Umweltverseucher. [Polychlorinated biphenyls as environmental contaminants.]** *Bundesgesundheitsblatt 15(19): 281; 1972.* (1 reference) (German)

Environmental hazards of PCBs are reviewed. Polychlorinated biphenyls are highly stable compounds which accumulate in the food chains of ecological systems. Correlation between the PCB level in the blood of sea birds and the rate of industrial PCB use was determined. Organochlorine pesticides potentiate the effects of PCBs; the latter have greater liver enlarging effects than DDT, DDE, and dieldrin. Human milk and adipose tissue samples investigated contained 3.5 ppm and 5.7 ppm PCBs, respectively. As no data on the possible hazards of PCBs is available, it is not yet possible to determine maximum allowable PCB concentrations in forage.

73-0513. Aebi, H. (Medizinisch-chemisches Institut, Bern, Switzerland). **Unser Schicksal: Mit Giften leben. [Our fate: living with toxicants.]** *Chem. Rundsch. 25(11): 283, 285, 287; 1972.* (22 references) (German)

General problems of environmental hazards, and of toxicants within the ecological systems in particular, are reviewed. Organochlorine pesticides have nonspecific action and accumulate in the organism, especially in adipose tissues, as a result of their being highly persistent. Unlike insects, mammals have a weak enzymatic detoxicating capacity for organochlorine pesticides. Efforts should be made to substitute other less harmful and less persistent preparations for these pesticides. However, it has not been possible to find adequate substitutes for several uses of DDT, especially for use in developing nations.

73-0514. Binder, E. (Abteilung Agrochemikalien, Emser Werke AG, Zurich, Switzerland). **Pflanzenschutzmittel-ja oder nein? [Pesticides–yes or no?]** *Chem. Rundsch. 25(31): 995; 1972.* (German)

General arguments for the use of pesticides are presented. Pesticides available on the market have been carefully tested by biologists, chemists, toxicologists, and medical doctors. The tolerance quantitites are worked out so carefully by experts that any danger to human health must be ruled out. However, to avoid large pesticide residue contents in crops, appropriate intervals between the last pesticide application and harvesting should be observed.

73-0515. Jukes, T. H. (Med. Physics, Univ. of California, Berkeley, CA). **An appraisal of recent developments in the regulatory situation of DDT.** *Clin. Toxicol. 5(3): 427-436; 1972.*

In the recently released report by the National Academy of Sciences, *Chlorinated Hydrocarbons in the Marine Environment*, marine biologists strongly opposed DDT, but the report drew heavily upon unpublished information and questionable extrapolations. In addition to the exaggeration of the testimony, newspapers and commentators altered the wording to create a more unfavorable image of DDT. Hearings were requested by the USDA and 30 DDT formulators trying to preserve the few remaining uses of DDT. For the first time, cross-examination provided a more balanced view of the subject. A number of witnesses testified for the Environmental Defense Fund and the Environmental Protection Agency, but in the opinion of the U.S. Surgeon General, the risk involved in the use of DDT for health purposes was not proven as endangering human life. The role of DDT in public health, especially in developing nations, has been impressive enough to want it available for continued use in the field.

73-0516. Anonymous. **Gesetze, Verordnungen, behoerdliche Verlautbarungen. [Laws, regulations, and official notices.]** *Deut. Lebensm.-Rundsch. 68(10): 333-335; 1972.* (German)

The DDT Law for West Germany and West Berlin, effective November 1972, is summarized. The production, import, export, sale, purchase, and use of DDT and its isomers as well as products containing these substances, are prohibited. The German Federal Public Health Administration may grant permission for the use of DDT and DDT products for research, investigation, and experimental purposes; for the synthesis of other products; as well as exclusively for the control of *Pediculidae, Monomorium pharaonis* and *Cimex lectularius* except in humans, vertebrates, stables, and localities used for the processing, handling, and selling of foodstuffs. The provisions of the July 23, 1971, bans and limitations concerning the use of plant protection products continue to be in force. Maximum allowable DDT residue contents in foodstuffs and other products for human needs will be determined.

73-0517. Anonymous. **"No intention of banning all pesticides."** *Farm Chem. Croplife 135(9): 26-27; 1972.*

The Environmental Protection Agency (EPA) considers all elements of society in its decisions on the pesticide question, and the decision is not just a scientific one. In the hearings on DDT two thirds of the testimony was from proponents of that insecticide. Reports of the hearings, particularly those in *Business Week*, contain errors regarding statements by witnesses which have misled or caused concern among farmers. The goal of EPA is to maximize the availability of pesticides according to the demand of the environment while minimizing the hazards of their use to society. The increased fees for pesticide petitions are not a step toward eliminating pesticides, but only a means of covering the increasing cost of processing such petitions.

73-0518. Anonymous. **Japan: emerging pesticide power.** *Farm Chem. Croplife 135(10): 36-65; 1972.*

Japan's pesticide industry has a favorable competitive position because of the low cost of development of new materials. Many new safer pesticides have been introduced there. Pesticides are responsible for Japan's tremendous gains in food production. Concern for the environment has resulted in the establishment of an environmental protection agency and increased requirements for pesticide registration and the restriction of mercurials, parathions, BHC, DDT, and some other organochlorines. A specially formulated microgranule (105 to 297 μm) was developed to solve the drift problem. The recently established Institute for Environmental Toxicology establishes safe limits for pesticide use and plans to operate on an international basis. A number of Japanese chemical companies have made significant contributions to the field. Mitsubishi has found amino acids and fatty acids promising as selective inhibitors of cell wall synthesis. A company-by-company listing of pesticides available from Japan is included.

73-0519. Schinzel, A. (Universitaet Innsbruck, Innsbruck, Austria). **Umwelteinfluesse auf die Qualitaet des Wassers. [Environmental influences on the water quality.]** *Gas Wasser Waerme 26(4): 66-70; 1972.* (German)

General problems of water pollution are reviewed with special regard to the effects of pesticides. The DDT concentrations in the Rhine, Main, and Danube water are 250 to 700 times lower than in plants and animals, and hence harmless for humans. Polychlorinated biphenyls interfere with enzyme and hormone metabolism. Carcinogenicity tests of DDT and similar pesticides have failed so far to yield unambiguous results. Mercury salt residues in water, having shown considerable increase for the past years in Austria, have a cumulation factor of 3,000 in fish and even more in fish liver and kidney. The maximum allowable mercury residue concentrations in water as determined in different countries range from 0.5 to 1 gamma.

73-0520. Anonymous. **Anwendungsverbote fuer bestimmte Pflanzenschutzmittel. [Ban on the use of certain pesticides for plant protection.]** *Gesunde Pflanz. 23(9): 157-160; 1971.* (German)

The July 29, 1971 order prohibiting or limiting the use of certain pesticides in West Germany is reproduced. The use of aldrin is permitted only for vineyard soil treatment to control *Otiorrhynchus sulcatus* F., and DDT may be used in forests for the control of *Hylobius abietis* L., *Rhyacionia buoliana* D. et Schiff and of *Choristoneura murinana* Hb, both till the end of 1974. Endrin may be used for the control of *Arvicola terrestris* L. in orchards until the same deadline, and so may heptachlor against soil insects in beet fields. The use of maleic acid anhydride is limited to non-agricultural areas. Mercury compounds may be used as seed treating agents for cereals, root crops except for potato, and for grasses. The use of lindane in granaries, of toxaphene in horticulture, and the distribution of 2,4,5-T from aircraft are prohibited.

73-0521. Leib, E. (Author address not given). **Pflanzenschutz ist mehr als Chemotherapie–Umweltschutz aus besonderer Sicht. [Plant protection is more than simply chemotherapy–special aspects of environmental protection.]** *Gesunde Pflanz. 23(11): 209-216; 1971.* (German)

The complex problem of plant protection is reviewed regarding environmental protection. It is stressed that the use of pesticides constitutes only one of the several possible methods of plant protection, as other non-chemical methods can be substituted for chemical ones. Plant hygiene, use of high-quality seed, proper choice of variety and soil, balanced nutrition, use of resistant varieties, use of biological enemies of the harmful organisms to be controlled, as well as the collection and destruction of pests and infested plant parts constitute many genuine, highly effective pest control methods more or less exempt from environmental hazards.

73-0522. Moore, N. W. (Toxic Chem. and Wildl. Sect., The Nature Conservancy, Monks Wood Exp. Station, Abbots Ripton, Huntingdonshire, England). **General review of ecological problems arising from pesticide usage in special sugar beet crops.** *IIRB Inst. Int. Rech. Betteravieres 5(4): 199-204; 1972.* (6 references)

Pesticides used on sugar beets, as on all crops, affect non-target species because they are nonspecific. The persistent organochlorines which are widely dispersed in the environment and concentrated in food chains are of particular concern to the conservationist, whose task is becoming one of management and control of renewable resources. Acute and sublethal effects attributed to these compounds have caused severe population declines of birds of prey in some areas. Many countries have subsequently prohibited or restricted the use of these chemicals. Little work has been done on the

effects on wildlife from the wide range of chemicals applied to sugar beets, but casualties have rarely been reported. Sugar-beet seed is sown at low densities and usually well-buried, also, sugar beet is not an attractive crop to birds at the time of year when most pesticides are applied. Although a hazard does not appear to exist now, future uses, if hazardous, should be modified so that minimal damage to wildlife is done while high crop yields are retained.

73-0523. Sharma, K. P.; Joshi, D. P.; Sekhon, K. S. (Dept. of Plant Breeding, Punjab Agr. Univ., Ludhiana, India). **Poisonous chemicals of common foodstuffs and their role in metabolism—a review.** *JNKVV (Jawaharlal Nehru Krishi Vishwa Vidyalaya) Res. J. 5(2): 66-76; 1971.* (175 references)

Poisonous substances obtained by consumption of plants as food which have been implicated in diseases and/or deaths are: pesticides and other agricultural chemicals containing arsenic or phosphorus; products of plant origin like oxalic acid, phytic acid, fluorine containing acids, senecio alkaloids, cyanogenic glycosides and certain amino acids; and substances caused by microbiologic infection in food such as aflatoxin. Parathion, the prototype of pesticides which are directly toxic, inhibits ChE. Organochlorines such as DDT, BHC, and dieldrin have been found in body fat. Poisoning by rodenticides of the anticoagulant type can be treated with vitamin K or by transfusion of whole blood. Several references are cited which deal with the effects of pesticides.

73-0524. Ostarhild, H. (Author address not given). **Die Pflanzenschutztechnik am Scheideweg. [Turning point in plant protection techniques.]** *Landtechnik 26(8): 213, 216-218; 1971.* (German)

General problems of plant protection and especially of spray equipment are described. Workmanlike use of spray equipment and of pesticides, including the observance of the normal dose, help prevent excessive residue contents in crops as well as poisoning hazards for both the equipment operator and the consumers. The problem of pesticide residue contents in food is relatively small in comparison with other environmental hazards such as air and water pollution and will be further lessened through the development of new, less hazardous products.

73-0525. Hillers, S. A. (Labor Red Banner Inst. of Organic Synthesis of the Latvian SSR Academy of Sciences, Riga, USSR). **O primenenii pestitsidov i merakh bor'by s zagryazneniyem okruzhayushchey sredy. [On the use of pesticides and on environmental protection measures.]** *Latv. PSR Zinat. Akad. Vestis 4: 34-43; 1972.* (15 references) (Russian)

General problems of the environmental hazards and of the determination of pesticides are reviewed. The use of pesticides accounts for about 2% of the total environmental pollution. While hazards involved in the acute effect of pesticides are expected to decrease as highly toxic pesticides are replaced by new products of high selectivity and low toxicity, chronic hazards continue to be prevalent. The DDT residue content in milk has been reduced to below 1% of the initial level since the use of DDT was prohibited five years earlier. Gas-liquid chromatography is the most valuable method for determining pesticide residues. A new fungicide, *N,N*-dimethyl-*N'*-phenyl-(*N'*-fluorodichloromethylthio)-sulfamide, is being tested. It has an oral toxicity of 1,000 mg/kg to rat and is eliminated from plants in 7-14 days.

73-0526. Andrusaitis, G. P. (Inst. of Biology of the Latvian SSR, Riga, USSR). **Vliyaniye pestitsidov na okruzhayushchuyu sredu. [Environmental effects of pesticides.]** *Latv. PSR Zinat. Akad. Vestis 4: 44-49; 1972.* (9 references) (Russian)

Environmental effects of pesticides and toxicological studies of 2,4-D are reviewed. Pesticides are suspected of accumulating in systems and triggering major shifts in the ecological equilibrium. The use of organochlorine pesticides such as DDT, aldrin, dieldrin, and endrin has been restricted in the USSR, the USA, France, Switzerland, Bulgaria, and Yugoslavia. Investigations show increasing DDT and PCB residue contents in the Baltic Sea. Flounder, sprat, salmon, and cod from the Baltic Sea contained 0.013-0.029%, 0.40-1.3%, 1.5-3.1%, and 0.21-0.32% of DDT and 0.021-0.056%, 0.27-1.0%, 0.3-0.54%, and 0.033-0.91% of PCBs on a fat basis. Experiments revealed the interference of 2,4-D with phosphorylation and oxidation processes in the mitochondria as well as swelling of the latter due to the destruction of the lipoprotein membrane structure. Reduction of the oxygen demand was observed in *Simocephalus vetulus* and *Misgurnus fossilis* embryo due to 2,4-D.

73-0527. Anonymous. **Des hormones, relais des insecticides. [Hormones as possible insecticides.]** *Nouv. Presse Med. 1(21): 1394-1395; 1972.* (7 references) (French)

Recent achievements in the development of new pest control methods are reviewed. Insect juvenile hormones synthesized recently are compounds with terpene-like structure. Such insect hormones are highly selective and nontoxic although unstable and expensive insecticides. Insecticides containing natural enemies such as *Bacillus thuringiensis*, of specific insects, are in practical use now. Products with genetic or sterilizing action are being developed.

73-0528. Stephan, F. (Abteilung fuer Obstbau, Gartenbau und Pflanzenschutz, Landwirtschaftskammer fuer Oberoesterreich, Austria). **Pflanzenschutz und Umweltschutz. [Plant protection and environmental protection.]** *Pflanzenarzt 25(5): 53; 1972.* (German)

General rules for the use of pesticides with respect to environmental protection and accident prevention are described. Pesticides in original packages should be used as instructed concerning concentrations and amounts needed and accident prevention rules. Pesticides should be used in periods of maximal effect as announced by pest warning and monitoring stations. Whenever possible, pest control should be performed in calm weather. The use of most organochlorine insecticides for fodder crops and that of certain insecticides such as aldrin and dieldrin in horticulture are prohibited.

73-0529. Unterstenhoefer, G. (Pflanzenschutz Anwendungstechnik, Biol. Forschung, Farbenfabriken Bayer AG, Leverkusen, Germany). **Integrated pest control from the aspect of industrial research on crop protection chemicals.** *Pflanzenschutz-Nachr. 23(4): 264-272; 1970.* (14 references)

The development of selective chemical pesticides as a possible contribution to integrated pest control is discussed. Monotoxic or strictly selective compounds are seldom discovered, extremely expensive to develop, and not economical to use. To use them successfully, an accurate diagnosis of the pest is needed. Often, they allow the expansion of other pest populations when used. Active ingredients of oligotoxic and polytoxic insecticides could be developed economically. Oligotoxic compounds, acting on a limited number of species, are most frequently discovered. They are selective in the sense of integrated control if activity does not extend to beneficial organisms, an unforeseeable chance occurrence. Use of the lowest possible concentration in a suitable formulation, application at the most favorable time, and the use of attractants to increase the probability of contact between active ingredient and pest can produce a certain amount of selectivity with the oligotoxic and polytoxic insecticides.

73-0530. Borlaug, N. E. (Author address not given). **Du mythe de l'equilibre naturel aux techniques modernes de l'agriculture. [From the mythical balance of nature to modern agricultural techniques.]** *Phytoma 24(241): 26-28; 1972.* (French)

Many views of environmentalists on the ecological effects of pesticides are criticized. The existence of a closed ecosystem in balance with nature is refuted on the basis of practical experience. Since the inherent regulatory function of nature is not sufficient, control by man with pesticides and fertilizers is necessary. While standard pesticides are satisfactory for 80-90% of all pest control problems, new control methods such as the use of parasites, resistant plant varieties, genetic and hormonal techniques, attractants and repellents, and of biodegradable insecticides should be developed.

73-0531. Planiere, J. (Author address not given). **Conference des Nations Unies sur l' environnement. [United Nations conference on the environment.]** *Phytoma 24(241): 37-39; 1972.* (French)

Resolutions adopted by the United Nations Conference on the Environment, held in Stockholm, June 1972, are reviewed. The resolutions concerning pesticides encompass studies on elimination and toxicity of pesticide residues, supervision of use in agriculture, the development of new pesticides with increased selectivity and innocuous residues, and ecouraging the use of thoroughly tested biological pest control techniques.

73-0532. Preston, Jr., W. H. (Pesticides Regul. Div., Environmental Protection Agency, Washington, DC 20460). **Comments on pesticides regulation activities of the Environmental Protection Agency.** *Proceedings of the 2nd Gulf Coast Conference on Mosquito Suppression and Wildlife Management, 1971, 17-19.*

The organization and officials of the Environmental Protection Agency (EPA) dealing with pesticides are presented; addresses of the Washington Headquarters and the 10 regional administrators are included. Current chemicals to be reviewed in either public hearings or an advisory committee include DDT, mirex, aldrin-dieldrin, 2,4,5-T, Dutch Elm Suppressant, and Lindane Vaporizer. Five years of surveillance has greatly reduced the contamination of pesticide products by organochlorines and organophosphates not included in the label. Dioxin levels in currently manufactured products have been reduced to a relatively safe level. EPA is looking at possible contamination of products with hexachlorodioxins and some related furans. Polychlorinated biphenyls are not known to be official inactive components of pesticide products at present. EPA is now reviewing the seriousness, extent, and persistence of ethylene thiourea. All of the information presented is subject to changes as the organization changes and as new information on pesticides is obtained.

73-0533. DeVaney, T. E. (Pesticides Regul. Div., Environmental Protection Agency, Washington, DC 20460). **Registration of pesticides–a review.** *Proceedings of the 2nd Gulf Coast Conference on Mosquito Suppression and Wildlife Management, 1971, 20-21.*

Many changes have been made in the pesticide registration process and data requirements for registration over the years. In 1947 the USDA was assigned registration responsibilities under the Federal Insecticide, Fungicide, and Rodenticide Act. The Act places the burden of proof of safety and efficacy on the manufacturer; a registration is effective for five years, but may be suspended at any time if necessary to protect the public interest. After 1963 review of applications was also done by the Departments of Health, Education and Welfare and the Interior when deemed necessary by the USDA. With the formation of the Environmental Protection Agency in 1970, personnel from each department who had been engaged in the registration process were

transferred to the new agency. The registration process has been improved and full consideration is given to environmental impact and the effects on fish and wildlife. Data requirements have been gradually increasing and emphasis has shifted from efficacy to toxicity and residues. New guidelines for studies to predict the environmental impact of pesticides were issued in 1969.

73-0534. Maugh, Jr., T. H. (Author address not given). **Polychlorinated biphenyls: still prevalent, but less of a problem.** *Science 178(4059): 388; 1972.*

Although polychlorinated biphenyls (PCBs) are ubiquitous and toxic to a variety of animals, they should decline in importance as environmental pollutants in the U.S. Wildlife from the Arctic Circle, water and bottom sediments from remote areas, a number of foods, and human adipose tissue have contained PCBs. Mink and many estuarine organisms are quite susceptible to these compounds. Hatchability of chicken eggs is drastically reduced by certain PCBs in the diet. New restrictions on disposal of PCBs and on their use in food containers, coupled with Monsanto's voluntary discontinuation of PCB sales that might allow the chemicals to enter the environment, should result in lower levels of contamination.

73-0535. Adler, C. (Electric Whale Company, 99 Nassau St., New York, NY 10038). **Falling watermelons.** *Science 178(4060): 450; 1972.* (5 references)

A Letter-to-the-Editor admonishes the scientific community not to stifle new ideas and new products because of fear of environmental damage. Determination of the full environmental impact of a man-made product would require vast numbers of biologist-person years and staggering costs; the probability of environmental danger from such things as DDT and polychlorinated biphenyls (PCB) is low. On the list of problems facing the world today, PCB contamination is near the bottom.

73-0536. Ramade, F. (Biologie Animale, Univ. de Paris-Sud, Paris, France). **Le mercure tue. [Mercury can kill.]** *Sci. Progr. Decouverte 3447(6F): 39-47; 1972.* (7 references) (French)

Findings concerning the accumulation of mercury, especially in the form of methylmercury, in the ecosystem, and toxicological investigations are reviewed. Mercury residues were determined at 28-150 mg/kg in the liver and kidney of pheasant and partridge, 8-45 mg/kg in pigeon, and 11-136 mg/kg in finch. The highest mercury content determined in granivorous birds was 270 mg/kg. Methylmercury contents of 10 ppm and 40 ppm were determined in diatoms and fish. The methylmercury content in chicken eggs in Sweden ranges from 27 to 29 ppb. Toxicological studies revealed teratogenic and antimitotic effects from methylmercury and phenylmercury.

73-0537. Mellanby, K. (Shell Ltd., Monks Wood Exp. Station, Huntingdon, England). **Averting world pollution.** *Span (London) 14(3): 136-138; 1971.*

An attempt is made to give an objective view on the pollution of our planet. Pesticides have caused concern in this area for some time. While they have had enormously beneficial effects in terms of food production and public health, they have damaged both man and his environment when improperly used. Much of this damage can be and is now being avoided by careful application of the chemicals. Most herbicides and many insecticides are not persistent and should not escape from the area of application. The persistent organochlorines cause the most concern, but use of these compounds has been successfully, voluntarily, curtailed in Britain and is being legislatively controlled in other countries in time to avert possible global harm. Present background levels do not produce any clinical symptoms in man, and partial recovery has been observed in species of wildlife affected by these compounds. Mercury, in the form of seed dressings, has not done serious ecological damage, but accumulation in the sea as a result of industrial dumping of mercury should be considered a potentially dangerous situation.

73-0538. Vierkoetter, S. (Abteilung Veterinaerwesen und Lebensmitteluebeberwachung, Senator Gesundheit und Umweltschutz, Berlin, Germany). **Untersuchung von Lebensmitteln auf gesundheitsgefaehrdende Rueckstaende. [Control of harmful residues in food products.]** *Ther. Gegenw. 111: 578-585; 1972.* (German)

Rulings concerning the use of pesticides and the control of pesticide and other residues in food products in West Germany are reviewed. The 1968 Pesticide Act requires demonstrated efficacy and toxicity control of all pesticides by the Federal Institute of Biology before their use is authorized. The use of DDT in food products has been prohibited by the 1971 DDT Act. Maximum allowable pesticide residue contents in fruits and vegetables have been established since 1966. A similar standard for the maximum allowable mercury content in seafood is being elaborated.

73-0539. Massing, H. (Lerchenstrasse 10, 4 Duesseldorf 11, Germany). **Die chemische Gewaesserunterhaltung in der wasserbehoerdlichen Praxis. [The water authorities practice in the maintenance of waters by chemical methods.]** *Wasser Boden 24(8): 237-239; 1972.* (12 references) (German)

Water management aspects of the use of aquatic herbicides are described with special regard to related regulations adopted in Lower Saxony. The use of herbicides for water weed control is to be reported to the water management authorities who in turn are required to prohibit use if damage is suspected. The use of herbicides in special protection zones, in mineral springs, and in water bodies used for drinking water supply is

prohibited as is use on dikes, in reservations and recreational areas, and in fishing waters. Water from herbicide-treated water bodies should not be used for irrigation during the first 14 days, for watering livestock during the first 10 days, and grass from such treated areas should not be used as or harvested for forage during the first 14 days. Containers of herbicides should bear such data as the name of the effective agent and recommended dosage.

73-0540. Sasse, K. (Wissenschaftliches Hauptlaboratorium der Farbenfabriken Bayer AG, 509 Leverkusen-Bayerwerk, Germany). **Chemical plant protection–past, present and future.** *Angew. Chem. Int. Ed. 11(4): 260-267; 1972.* (14 references)

Chemical control of crop diseases contributes immensely to not only the nutritional but also the economic and political well-being of a country. The breakthrough in the development of chemical plant protection was the introdution of DDT in 1940, followed by the introduction of the organophosphate parathion in 1944. Alkyl- and arylmercury compounds have broad biological activity as seed dressings. Recently discovered systemic fungicides confer an internal prophylaxis on the plants, independent of stability of the surface residues, and have made possible control of rust and loose smut for the first time. The most important herbicides are the phytohormone-type which kill virtually only dicotyledons and can be used on cereals. Although problems of residues and the emergence of resistance suggest the need for non-chemical or integrated control programs, little success has been achieved in this area. Most countries have established tolerances for chemical agents in food designed to prevent abuse, but these vary. At present Germany has declared a zero tolerance for arsenic compounds, fluoroacetic acid and its derivatives, mercurials and selenium, and ten individual agents. Chemicals will continue to be important for plant protection; international laws should be formulated concerning their use.

73-0541. Koehler, E. (Landwirtschaftl. Versuchsstat., Badische Anilin- und Soda-Fabrik AG, Limburgerhof, Germany). **Gegenwart und Zukunft des chemischen Pflanzenschutzes aus industrieller Sicht. [Present and future prospects of chemical plant protection from the industrial viewpoint.]** *Z. Pflanzenkr. Pflanzenphysiol. Pflanzenschutz 79(3): 152-159; 1972.* (8 references) (German)

The world wide consumption of pesticides increased from about 2 billion German marks' worth in 1955 to 3 billion in 1960, 5.5 billion in 1965, and 11 billion in 1970. If the trend continues, total consumption by 1980 will have a value of 30 billion marks. However, the future rise is not expected to be as rapid as in the past because of the desire to prevent environmental pollution and the resulting tightening of testing regulations. Biological-control growth is expected to accelerate as chemical use tapers off. The need for chemical pesticides in large quantities is partially sustained by the progressive mechanization of agriculture. The high competitive risks and small profit margins for farmers and pesticide manufactures will continue to apply pressure in favor of the most economically efficient plant-protection means. Research and development costs are about 5-10% of the total cost of pesticide use, not counting the cost of basic research performed by universities and government-sponsored institutions.

73-0542. Bourke, J. B.; Loftus, G.; Lisk, D. J. (Pestic. Residue Res. Lab., N.Y. State Agr. Exp. Sta., Geneva, NY 14850). **A pesticide residue data information retrieval system.** *J. Agr. Food Chem. 20(6): 1275-1278; 1972.* (4 references)

The retrieval of laboratory, residue, and field data necessary for the formulation of safe and effective pest control programs is accomplished by a computer search of previously coded data collected from various locations. The retrieval program uses up to 16 search keys, including chemical, crop, and analytical method used. A printed report is prepared which contains 24 field and laboratory parameters, sample identification number, and residue found. The program is designed to run on a small computer using disk storage for all data. (Author abstract reprinted by permission of the American Chemical Society)

73-0543. Fink, R. J. (Dept. of Agr., Western Illinois Univ., Macomb, IL). **Phytotoxicity of herbicide residues in soils.** *Agron. J. 64(6): 804-805; 1972.* (5 references)

Relative dissipation rates and carryover phytotoxicity of three triazine herbicides were compared using oats, soybeans, or alfalfa planted in soil taken at 6, 12, 18 and 26 wk after fields were planted with corn and treated. All pre- and post-emergent atrazine and cyprazine treatments (except cyprazine at 1.12 kg/ha) sampled 26 wk after treatment significantly reduced alfalfa yields. Alfalfa yields were reduced in soil treated with 2-(4-chloro-6-ethylamino-*s*-triazine-2-ylamino)-2-methylpropionitrile (SD-15418) when sampled at 6 or 12 wk but not at 18 or 26 wk. No effect was observed with alachlor-treated soils on alfalfa, oats, or soybeans. Oats grown in soils collected at 26 wk were not affected by SD-15418 residues, but serious injury was caused in oats by cyprazine and atrazine residues (2.8 kg/ha application) at all sample times. Post-emergence applications of these chemicals had a greater effect because of the 20 day delay and larger residues. Atrazine and cyprazine reduced yields in soybeans when soils were sampled at 6 or 12 wk after treatment. These studies indicate that the order of herbicide residual level in the soil was cyprazine $\geqslant$ atrazine, followed by SD-15418, and then alachlor. Growers can probably eliminate carryover injury to susceptible crops by using short-residual herbicides such as alachlor or reducing amounts of more persistent chemicals and combining them with effective short-residual ones.

73-0544. Cecil, L. K. (Tucson, AZ). **Difficult pollution control problems.** *A.I.Ch.E. Ind. Process Des. Water Pollut. Contr., Proc. Workshop, 3rd: 1970, pp. 99-102.*

Among the numerous engineering problems involved in reducing pollution economically is that of treating waste water to remove small quantities of DDT. The Federal Water Pollution Control Administration is planning to enter negotiations with a private company for investigating means for controlled self-destruction of pesticides. Perhaps in the future DDT may be formulated with a chemical capable of degrading it to prevent its accumulation in the aqueous environment.

73-0545. Chiba, M.; Doornbos, F. (Res. Station, Canada Dept. of Agr., Vineland Station, Ontario, Canada). **Studies on the degradation of DDT during fermentation of grapes and its solubility in wine.** *Amer. J. Enol. Viticult. 22(4): 189-193; 1971.* (25 references)

The maximum average DDT residue found in four cultivars of grapes tested was 0.87 ppm. In wines made from the four cultivars of grapes the highest average DDT residue at 1 mo was 3.9 ppb and after 4 mo no detectable residue remained in this wine. No correlation was observed between the residues in the grapes and the wines. Wine made from grapes to which 50 ppm DDT was added also had no detectable (0.2 ppb) residues after four mo. Six commercial wines contained no trace of DDT. Batches fortified with labeled DDT contained increasing TDE levels in 5-day and 1-mo samples of lees (filtered material). TDE was not detectable in the wine. DDE levels stayed constant in the lees and were not found in the wine. Conversion of DDT to DDA was noted in unfiltered wine and filtered wine kept in cold storage, and it was removed in the lees. Alcohol concentration did not seem to affect the level of DDT residues.

73-0546. Coic, Y.; Tendille, C. (Station Centrale de Physiologie Vegetale, C.N.R.A., Versailles, France). **Causes connues des variations quantitatives des oligo-elements dans les vegetaux. [Known factors influencing the trace element contents in plants.]** *Ann. Nutr. Aliment. 25: B97-131; 1971.* (125 references) (French)

Various factors known to influence the quantitative variations of trace elements in plants are reviewed. The copper, manganese, and zinc contents in plants can be influenced by the application of fungicides such as organic copper compounds, maneb, mancozeb, zineb, and ziram containing trace elements. While these fungicides are of low solubility, the plants can utilize manganese present in maneb as well as zinc present in zineb and ziram. Organic copper compounds, especially when applied in large amounts, are phytotoxic.

73-0547. Wagenbreth, D.; Kluge, E. (Abt. fuer Forstsch. gegen Pflanzl. Schaedlinge, Inst. fuer Forstwiss. Eberswalde, Akad. der Landwirtschaftswiss. zu Berlin, DDR). **Der Einfluss von Fungiziden (Thiram, Vapam) auf den Triazinabbau im Boden. [The effect of fungicides (thiram, Vapam) on triazine degradation in the soil.]** *Arch. Pflanzenschutz 7(6): 451-459; 1971.* (17 references) (German)

Triazine herbicides are thought to undergo microbial degradation in soil, where soil fungi and actinomycetes contribute heavily. To test the effects of soil fungicides on triazine degradation, pots of soil mixed with prometryne, atrazine, or simazine were treated with thiram or Vapam (metham) and incubated in the laboratory. Soil samples were taken periodically for chemical determination of fungicides and herbicides, as well as for bioassays with oats and *Rhizoctonia*, a soil fungus. Degradation of thiram appeared essentially complete by the chemical tests within 30 days while slight activity was detected in the bioassay for more than 127 days. Although degradation of prometryne was essentially complete within 30 days in the pots of soil without fungicide, complete degradation (by both chemical and biological criteria) took 195 days in the thiram-treated soil. Results with Vapam were similar to those with thiram. Little degradation of atrazine or simazine took place during the experimental period in the control pots, and degradation in fungicide-treated pots was even slower. The lag period in the prometryne degradation curve was prolonged beyond the time at which thiram could still

be detected in the soil; this could be due to microbial antagonism or to a delayed regrowth of the fungal flora.

73-0548. Ebing, W.; Schuphan, I. (Inst. fuer Pflanzenschutzmittelforsch. der Biol. Bundesanstalt fuer Land- und Forstwirtsch., Berlin-Dahlem, Germany). **Umwandlungsprodukte von Pestiziden als umweltbelastende Stoffe. [Pesticide degradation products as environmental pollutants.]** *Ber. Landwirt. 50(2): 325-347; 1972.* (290 references) (German)

The study of pesticide residue degradation products, their interactions, and their environmental effects is relatively untouched. Despite the volume of literature already published on pesticide degradation, much more research is needed. Information is needed to resolve the purely conjectural areas present in most degradation schemes and to resolve unanswered questions such as residue interactions. Most published reports are based on recovery of less than half of the original quantity of pesticide; the fate of the remainder is unknown. Specific groups of pesticide degradation products reviewed here include phosphoric acids and derivatives, dehalogenated compounds, dealkylated compounds, epoxides and clathrates of chlorinated cyclodiene insecticides, aliphatic hydroxyl compounds, phenols, carbonyl compounds, sulfoxides and sulfones, nitrogen-containing heterocyclic metabolites, nitrogen-containing non-heterocyclic metabolites, mercaptans, isothiocyanates, thioureas, thiurams, carbon disulfide, carbon dioxide, and conjugates.

73-0549. Rebmann, H. (Milchwirtschaftl. Forsch.- und Untersuch. GmbH, 2 Hamburg, Germany). **Moeglichkeiten zur Entfernung von Insektizidrueckstaenden aus Futtermitteln. [Possibilities for removal of insecticide residues from feeds.]** *Ber. Landwirt. 50(2): 359-370; 1972.* (66 references) (German)

Some types of animal feeds contain substantial quantities of pesticide residues which are undesirable from the viewpoint of toxicity to animals as well as possible introduction into human foods. The removal of residues from feeds depends on the chemical structure of the compounds as well as the age and distribution of the residues. Decomposition and removal of insecticides is generally promoted by high-temperature drying, washing, extraction with water or organic solvents and vapors, or prolonged moist storage. Specific decontamination means suited to certain pesticides and crops are reviewed; for example, 20-90% of parathion residues can be removed from lettuce and celery by washing with alkaline hydrogen peroxide solutions. Ensilation of hay appreciably enhances the degradation of DDT residues whereas endosulfan residues are scarely affected. In one study seed wheat containing malathion was treated with nucleophilic reagents such as calcium polysufide for 40-80 min at 25° and the malathion content reduced from 500 or 1000 to 1 ppm. The reduction of insecticide content in the butterfat during buttermaking suggests that some organochlorines are bound to phospholipids and removed in the buttermilk. Irradiation, adsorption, and molecular distillation are other means of residue removal.

73-0550. Kohsiek, H. (Inst. fuer Anwendungstechn. der Biol. Bundesanstalt fuer Land- und Forstwirtsch., 33 Braunschweig, Germany). **Beifrag der Anwendungstechnik zur Minderung der Umweltbelastung mit Pestiziden. [Contribution of application technology to the reduction of environmental pollution by pesticides.]** *Ber. Landwirt. 50(2): 371-382; 1972.* (150 references) (German)

Pesticide application technology is concerned with improving uniformity of application, preventing drift, and using the smallest quantities of pesticides and carriers necessary to attain the desired results. Research efforts are directed toward developing improved apparatus, techniques, and chemicals, and adapting each of these to suit the others. Pesticide deposition study methods are continuously reevaluated, especially with respect to standardization of test conditions. Pesticide contamination of untreated areas primarily results from drift of spray particles, or possibly of dust containing adsorbed pesticides, but may also be due to evaporation from treated surfaces and codistillation with water vapor from plants and soil. Thus every pesticide-treated surface represents a potential source of atmospheric contamination. Attempts to reduce drift have been based on changing the physical characteristics of pesticides and selecting certain particle size ranges.

73-0551. Tolle, A.; Heeschen, W.; Bluethgen, A. (Inst. fuer Hyg. der Bundesanstalt fuer Milchforsch., 23 Kiel, Germany). **Chlorierte Insektizide, Fasziolizide und Antibiotika in der Milch. [Chlorinated insecticides, fasciolicides, and antibiotics in milk.]** *Ber. Landwirt. 50(3): 720-733; 1972.* (21 references) (German)

To provide a first, comprehensive set of statistics on chlorinated insecticide residues in West Germany, 405 milk samples, 748 butter samples, and 1147 cheese samples from 14 milk collection districts were analyzed in 1970/71. The values obtained were compared with the practical residue limits (PRL) established by the FAO/WHO. Weighted averages for all samples tested revealed 0.15 ppm α-BHC, 0.05 ppm lindane, 0.03 ppm heptachlor, 0.03 ppm aldrin, 0.05 ppm heptachlor epoxide, 0.11 ppm DDT + dieldrin, and 0.3 ppm total DDT. Butter samples exceeded the PRL more frequently than milk or cheese samples. Insecticides ingested with one liter of milk would account for 0.2% of the PRL for lindane, 7.7% of the PRL for heptachlor, 33% of the PRL for aldrin/dieldrin, and 6.7% of the PRL for DDT, on the average. Allowing for the consumption of various dietary components in Germany, the DDT consumption via milk fat would account for 45% of the daily DDT

intake. This contrasts with the 15% in the U.S. where DDT contents of other dietary components are higher than in Germany. The use of chlorinated hydrocarbons on fodder stands must be avoided and the chlorinated hydrocarbon contents of imported feeds monitored. Methods for preventing excessive residues of fasciolicides and antibiotics in milk are also discussed.

73-0552. Jung, N.; Mattheis, I.; Arfert, H. (Bezirksnaturschutzverwaltung, Rat des Bezirkes Neubrandenburg, Nonnenmuehle, DDR). **Zur Kontamination von DDT und HCH in Wildvogeleiern in der DDR. [Contamination of wild-fowl eggs with DDT and BHC in East Germany.]** *Biol. Rundsch. 9(5): 335-337; 1971.* (27 references) (German)

Residue contents of DDT and BHC were determined by thin-layer chromatography in 133 eggs of *Anas platyrhynchos, Anser anser, Buteo buteo, Fulica atra, Vanellus vanellus, Larus ridibundus, Cuculus canorus, Ficedula hypoleucos, Turdus merula, Parus major, P. ater, P. cristatus* and *Corvus corone cornix*, collected from 44 nests in various biotopes. Two eggs of *Anas platyrhynchos* and of *Buteo buteo* contained 0.69 ppm and traces of BHC, respectively, while DDT/DDE was found in all eggs of all but one species (*Vanellus vanellus*). The average DDT/DDE residues ranged from 1.12 to 6.75 ppm, with minimum and maximum of 0.43 and 25.3 ppm, respectively. The rate of DDT metabolism was 54.2%. The findings show relatively high degree of contamination from DDT and increased DDT residue contents in eggs of zoophagous species as compared with those of omnivorous and phytophagous species.

73-0553. Richou-Bac, L.; Mollet, M.-F.; Pantaleon, J. (Service d'Hygiene Alimentaire, Lab. Central de Recherches Vet. d'Alfort, Maisons-Alfort, France). **Etat actuel de la contamination des produits laitiers par les residus de pesticides organo-chlores. [Actual state of the contamination of dairy products with organochlorine pesticides.]** *Bull. Acad. Vet. Fr. 45(3): 131-146; 1972.* (11 references) (French)

Organochlorine pesticides in milk products were determined and compared with data from previous years. The total BHC residue content has gradually decreased from 0.92 ppm in 1967 to 0.29 ppm in 1971. Lindane residues show little variation and hexachlorobenzene concentrations are very low throughout. The heptachlor epoxide and heptachlor residue contents have increased from 0.02 ppm in 1967 to 0.10 ppm in 1971 while the dieldrin and aldrin residues have changed little during the same period (from 0.02 ppm to 0.09 ppm). The residue concentration of DDT and its metabolites decreased from 0.02 ppm in 1968 to less than 0.001 ppm in 1969.

73-0554. Monod, J.-L.; Raybaud, H.; Venot, C.; Carrara, G. (Station Marine d'Endoume, Marseille, France). **Identification et dosage des polychlorobiphenyles (P.C.B.) et des metabolites du D.D.T. chez la sardine (*Sardina pilchardus* L.).[Identification and determination of PCB and DDT metabolite residues in *Sardina pilchardus* L.]** *Bull. Soc. Pharm. Marseille 20(75): 155-160; 1971.* (20 references) (French)

A gas-chromatographic method for the determination of PCBs and DDT metabolites in lipids and the results of residue determination in 15 specimens of *Sardina pilchardus L.* caught in the Bay of Marseille are described. Lipids are extracted for determination in a Soxhlet apparatus by means of hexane and acetone used in a ratio of 2:1 as solvents; the extract thus obtained is evaporated under vacuum. Nitrogen is used as the carrier gas for gas-chromatographic determination; the column and injection temperatures are 200 and 220 C, respectively. The *p,p'*-DDE, *p,p'*-DDD (TDE) plus *p,p'*-DDT, total DDT, and PCB residues ranged from 0.1-1.07, 0.11-0.95, 0.22-1.73 and 0.7-5.1 ppm respectively. The results indicate dangerously high PCB concentrations in *Sardina pilchardus* L.

73-0555. Ceribashi, M.; Marga, K. (Lab. i Toksikol. Ushqimore Prane Inst. to Higj. dhe Epidemiol., Tirana, Albania). **Percaktimi i mbetjes se insekticidit vofatoks ne rrush. [Determination of Wofatox insecticide residues in grapes.]** *Bul. Shkencave Bujqesore 3: 117-122; 1971.* (9 references) (Albanian)

In tests on the persistence of Wofatox (methyl parathion) residues on grapes, vineyards were sprayed with 0.1, 0.15, and 0.2% solutions of Wofatox. The Wofatox residues dropped to 1 ppm within 10-25 days after spraying. To eliminate the possibility of excessive Wofatox residues in grapes at harvest time, the last spraying should be performed at least 20 days before harvest. Wofatox should be used at the lowest possible concentration, 0.1-0.15%. The solution should be agitated constantly during spraying to provide for uniform distribution of the active ingredient. Periodic analyses of local produce should be made to ensure that the Wofatox residues do not exceed 0.5-1 ppm.

73-0556. Derache, R. (Groupe de Recherches sur la Toxicologie des Aliments et des Boissons, INSERM, Toulouse, France). **La biodegradation ecologique des pesticides et le risque de pollution des aliments. [Biodegradation of pesticides in the ecology and the risk of food contamination.]** *Cah. Nutr. Diet. 7(2): 125-137; 1972.* (25 references) (French)

Possible degradation processes of pesticides occurring in water, air, soil, plants, and animals are reviewed. While both air and water may serve as vehicles for pesticides, physicochemical reactions such as hydrolysis and photolysis taking place in these media are the major cause of degradation, especially of organophosphorus pesticides. Pesticides such as simazine, triazines, dalapon, and diuron are decomposed by the soil microflora. Residues, primarily of organochlorine pesti-

cides, accumulate in adipose tissues. Pesticides can be metabolized in water, soil, plants, and animals.

73-0557. Kadis, V. W.; Yarish, W.; Molberg, E. S.; Smith, A. E. (Alberta Dept. of Agr., Dairy and Food Lab., Edmonton, Alberta, Canada). **Trichloroacetic acid residues in cereals and flax.** *Can. J. Plant Sci. 52(4): 674-676; 1972.* (2 references)

Trichloroacetic acid (TCA) residues in grains were determined by decomposing the TCA to chloroform and collecting it in toluene or pyridine for gas chromatographic or colorimetric analysis. Wheat, barley, oats, and flax grown on test plots were sprayed at the two- to three-leaf stage with sodium TCA; forage samples were harvested at 45 days and the seed collected from the crops at maturity. The mature grains contained from 0.0 to 2.4 ppm TCA and forage contained from 3 to 65 ppm. The results were within the same range for the various grains, test plot locations, and years. However, a decrease in residues on forage collected in Lacombe, Alberta, in 1971 compared to samples harvested in 1970 was attributed to climatic differences between the growing seasons.

73-0558. Nagl, H. G.; Korte, F. (Organisch-Chemisches Inst., Universitaet Bonn, Bonn, Germany). **Beitraege zur oekologischen Chemie 47. Reaktionen von Dieldrin mit Stickstoffdioxyd und Ozon in ultraviolettem Licht. [Contributions to ecological chemistry No. 47. Reactions of dieldrin with nitrogen dioxide and ozone in UV light.]** *Chemosphere 1(4): 143-146; 1972.* (8 references) (German)

Reactions of dieldrin with nitrogen dioxide and ozone present in small concentrations were studied in ultraviolet light with wave lengths larger than 300 nm. Dieldrin formed eight different metabolites in a two-stage reaction with nitrogen dioxide or ozone. The initial reaction was due to oxygen attack on the double bond or to hydrogen removal at the C-7 or C-2 atom. The formation of the nitro compound or nitrite from the *tert*-alkyl radical and nitrogen dioxide were observed as were those of the alkoxyl radical from the alkyl radical and ozone in secondary reactions. The alkoxyl radical reacts with R-H to form the hydroxyl compound, or with nitrogen dioxide to form nitrate.

73-0559. Moza, P.; Weisgerber, I.; Klein, W. (Inst. fuer oekologische Chemie, Gesellschaft fuer Strahlen und Umweltforschung, Munich, Germany). **Beitraege zur oekologischen Chemie 50. Auswaschen eines wasserloeslichen Aldrin-^{14}C-Abbauprodukts aus Boden. [Contributions to ecological chemistry No. 50. Leaching of a water-soluble degradation product of ^{14}C-aldrin from soil.]** *Chemosphere 1(5): 191-195; 1972.* (12 references) (German)

Leaching of a water-soluble metabolite of ^{14}C-labeled aldrin introduced into boxed soil at 10 cm depth was studied over three years. Ten per cent of the aldrin was leached out in three years. The metabolite found in the leachate was more hydrophilic than aldrin or its known metabolites; it was identified as dihydrochlordene dicarbonic acid dimethylester. The same metabolite was also found in soil and plants grown in the aldrin-treated soil. The soil samples investigated also contained another even more hydrophilic metabolite not yet identified.

73-0560. Kubacka-Szmidtgal, M. (Institute of Sugar Industry, Warsaw, Poland). **Badania pozostalosci pestycydow w surowcu buraczanym. [Investigation of pesticide residues in sugar beet.]** *Gazeta Cukrow. 80(9): 231-234; 1972.* (5 references) (Polish)

Soil in experimental fields contained 0.873 ppm BHC and 0.02138 ppm DDT residue five years after last application of pesticides. Determination of residues in sugar beets three months after single application of Tritox (10% DDT, 4% methoxychlor, 0.5% lindane) at 500 g/100 l. H_2O × 5 l./ha or of Ditox (5% DDT and 0.3% lindane) at 30 kg/ha revealed 0.05 ppm BHC and 0.37 ppm DDT in leaves and 0.0040 ppm BHC and 0.0182 DDT in roots of the plant. The amount of pesticide residue in leaves increased from that determined six weeks earlier (from 0.018 ppm BHC and 0.17 ppm DDT), suggesting that sugar beets absorb and accumulate pesticides in their tissues during the entire period of growth. The substantially larger amounts of BHC and DDT (11 and 20 times respectively) in leaves than in roots is probably related to the amount of lipids accumulating these compounds. The amount of BHC decreased during the three months' period (0.80 ppm in fields treated with Tritox, 0.83 ppm in those treated with Ditox) while the amount of DDT increased by 0.15 and 0.11 ppm, respectively, indicating the greater persistence and accumulation of this compound.

73-0561. Wagner, F.; Staerk, H.; Suess, A. (Pflanzenschutzberatung, Regierung von Niederbayern, Germany). **Veraenderung des Bromgehaltes in der Pflanze nach einer Bodenentseuchung mit Methylbromid. [Changes in the bromine levels in plants following soil treatment with methyl bromide.]** *Gesunde Pflanz. 23(11): 221-223; 1971.* (5 references) (German)

Bromine levels in radish, onion, beetroot, paprika, cucumber, bean, turnip, celery, and other vegetables were determined 3 and 6 months after soil was treated with methyl bromide in May of the same year. The bromine levels in onion, beetroot, and bean determined 6 months after the application of methyl bromide were considerably lower those found 3 months earlier; the bromine residue content in paprika was fairly constant during the same period. The bromine residue levels in radish and cucumber increased with the time. The bromine levels of vegetables grown in the same soil in the following year were higher than the background and the maximum allowable contents.

73-0562. Rose, F. L.; McIntire, C. D. (Battelle-Northwest, Richland, WA). **Accumulation of dieldrin by benthic algae in laboratory streams.** *Hydrobiologia 35(3-4): 481-493; 1970.* (13 references)

The effects of current velocity, light intensity, and species composition on the accumulation of dieldrin by benthic algal communities were studied in twelve enclosed streams receiving water from a natural creek. HEOD (dieldrin) concentrations ranging from 0.05 to 7.0 ppb were maintained in the water of these streams for 2-4 months; the water in the control stream was always less than 0.05 ppb HEOD. Analyses of algal samples by gas chromatography showed dieldrin concentrations ranging from 0.1 to 200 ppm; these results indicated that increases in concentration up to 30,000 times that of the water had occurred. Measurements taken with current velocities at zero and 28 cm/sec and with shaded and unshaded streams showed no significant differences in algal concentrations directly related to these physical factors. Quantitative evaluation of species composition revealed that communities dominated by filamentous algae accumulated greater amounts of the insecticide than those in which unicellular diatoms were prevalent. The mechanisms responsible for dieldrin concentration by algae were not clearly indicated in these studies.

73-0563. Chopra, S. L.; Girdhar, K. C. (Dept. of Chem. and Biochem., Punjab Agr. Univ., Ludhiana, India). **Persistence of malathion *S*-1,2-bis(ethoxy carboxyl)ethyl *O,O* dimethyl phosphorodithionate in Punjab soils.** *Indian J. Appl. Chem. 34(5): 201-207; 1971.* (7 references)

The degradation of malathion in three types of Indian soil increased with its increase in concentration. Malathion was decomposed more rapidly in alkaline than in acidic soil. Time studies indicated that complete degradation took place in the soils studied within four days and exposure to UV light reduced the time for complete degradation to 10 hr. Increasing temperature and/or humidity also enhanced the rate of decomposition. The reaction was slowest at neutral pH and degradation followed a first order reaction.

73-0564. Miller-Ben-Shaul, D. (Jerusalem Biblical Zoo, Jerusalem, Israel). **Residual organochlorine insecticides in the body fat of zoo animals and in foods fed to zoo animals.** *Int. Zoo Yearb. 11: 236-238; 1971.* (1 reference)

Peritoneal fat tissue of zoo animals which died from various causes contained high levels of total organochlorine insecticide (total DDT). Values ranged from 0.9 ppm in the budgerigar to 36.1 in the hedgehog. Results of food analyses indicated that carnivores and birds of prey consumed by far the largest amount of organochlorines per kilogram of body weight. None of the food except mice and locusts was grown specifically for animal consumption. Whole fish contained an average of 47.3 ppm and whole cooked chicken an average of 16.4 ppm. Average alfalfa residues were as low as 0.4 ppm. Animals dying during their acclimatization periods could have high organochlorine blood levels. Studies are underway to determine the effect of intake of these insecticides on reproduction of birds and mammals.

73-0565. Causey, K.; McIntyre, Jr., S. C.; Richburg, R. W. (Dept. of Zool. Entomol., Auburn Univ., Auburn, AL). **Organochlorine insecticide residues in quail, rabbits, and deer from selected Alabama soybean fields.** *J. Agr. Food Chem. 20(6): 1205-1209; 1972.* (16 references)

Samples of edible meat were collected from bobwhite quail (*Colinus virginianus*), swamp and cottontail rabbits (*Sylvilagus aquaticus* and *S. floridanus*), and white-tailed deer (*Odocoileus virginianus*) found in or adjacent to selected soybean fields in Alabama and from areas with little or no history of insecticide use. Samples were analyzed for residues of organochlorine and organophosphate insecticides. DDT and its metabolites (DDT + DDE + DDD (TDE)) were the only insecticides occurring consistently in the samples tested. Toxaphene, heptachlor epoxide, and dieldrin were detected in a small percentage of the animals. Total DDT residues (DDT + DDE + DDD reported on a lipid basis) averaged 17.08 ±, 3.00±, and 2.47± ppm, respectively, for bobwhite quail, white-tailed deer, and rabbits collected from treated soybean fields. Total DDT residues averaged 1.68±, 0.10±, and 0.05 ± ppm, respectively, for bobwhite quail, white-tailed deer, and rabbits collected from areas with little or no history of insecticide application. (Author abstract reprinted by permission of the American Chemical Society)

73-0566. Hilton, H. W.; Robison, W. H. (Experiment Station, Hawaiian Sugar Planters' Ass., Honolulu, HI 96822). **Fate of zinc phosphide and phosphine in the soil-water environment.** *J. Agr. Food Chem. 20(6): 1209-1213; 1972.* (7 references)

Zinc phosphide, mixed with three soils at five moisture levels, decomposed with the liberation of variable amounts of phosphine gas. Oxidation at the soil surface yielded zinc and phosphate ions as the ultimate products. In a closed system the soils slowly reabsorbed and oxidized the PH_3. Phosphine placed at high concentration in contact with the same soils oxidized slowly and incompletely to phosphate ion. The rate of zinc phosphide decomposition increased with increasing moisture; phosphine absorption decreased with added water. Soil types differed markedly in the ability to oxidize Zn_3P_2 or PH_3. The differences could not easily be related to known soil properties. Zinc phosphide did not decompose in water from streams, domestic source, or the ocean. Acids and bases hydrolyzed Zn_3P_2 to PH_3 but the reaction was not entirely pH dependent. A

phosphate buffer of pH 7.00 extensively hydrolyzed Zn_3P_2 at room temperature. (Author abstract reprinted by permission of the American Chemical Society)

73-0567. Saltzman, S.; Kliger, L.; Yaron, B.* (Inst. of Soils and Water, Agr. Res. Organization, Volcani Cent., P.O.B. 6, Bet Dagan, Israel). **Adsorption-desorption of parathion as affected by soil organic matter.** *J. Agr. Food Chem. 20(6): 1224-1226; 1972.* (7 references)

Influence of soil organic matter on the adsorption-desorption of parathion was studied by using tagged insecticide. It was found that the parathion adsorption by soils is dependent on the type of association between the organic and mineral colloids. In aqueous solutions the parathion has a greater affinity for organic than for mineral adsorptive surfaces. Parathion bonding is stronger on organic than on mineral surfaces. (Author abstract reprinted by permission of the American Chemical Society)

73-0568. Norman, S. M.; Fouse, D. C.; Craft, C. C. (Agr. Res. Service, U. S. Dept. of Agr., Pomona, CA 91769). **Thiabendazole residues on and in citrus.** *J. Agr. Food Chem. 20(6): 1227-1230; 1972.* (12 references)

Citrus fruits coated with wax containing thiabendazole were assayed for thiabendazole by two procedures to determine what factors might affect methodology and quantitative reliability. Some factors studied were sampling, interference by citrus constituents, assay by surface stripping and by extracting ground whole fruit, spectrophotofluorometric and spectrophotometric measurements, commercial application, and experimental storage. Thin-layer chromatography eliminated citrus constituents that occasionally interfered with measurements of less than 1 ppm of thiabendazole. Surface stripping provided significantly higher residues than extracting ground whole fruit. Spectrophotometric and spectrophotofluorometric measurements were comparable; however, spectrophotofluorometric measurements were more sensitive. Thiabendazole residues varied two- to threefold from fruit to fruit with commercial application of thiabendazole in wax. Foam washing of lemons after storage removed 50-75% of the thiabendazole applied in wax before storage. Thiabendazole residues apparently decreased during storage. (Author abstract reprinted by permission of the American Chemical Society)

73-0569. Koeman, J. H.; Pennings, J. H.; De Goeij, J. J. M.; Tjioe, P. S.; Olindo, P. M.; Hopcraft, J. (Inst. of Vet. Pharmacol. and Toxicol., Utrecht, The Netherlands). **A preliminary survey of the possible contamination of Lake Nakuru in Kenya with some metals and chlorinated hydrocarbon pesticides.** *J. Appl. Ecol. 9(2): 411-416; 1972.* (17 references)

Birds collected in November, 1970, around Lake Nakuru in Kenya contained trace amounts of DDT, DDE, and dieldrin. Levels of endrin and TDE were below the limit of detection. Fish regurgitated by cormorants or found in the gut of the birds also contained extremely low levels of organochlorines. This may indicate that the rate of disappearance of the organochlorines is relatively high in these tropical regions. Tissue level determinations of several metals and pathological examinations were also carried out.

73-0570. Hanyu, A.; Takahashi, J. (Tochigi Prefect. Agr. Exp. Sta., Utsunomiya, Tochigi Prefect., Japan). **[Uptake of organochlorine insecticides by several crop plants.]** *Kanto Tosan Byogaichu Kenkyukai Nenpo (Proc. Kanto-Tosan Plant Prot. Soc.) 19: 123; 1972.* (Japanese)

Field applications of several organochlorine pesticides (BHC, DDT, aldrin, heptachlor, endrin, and dieldrin) to a variety of vegetable and fruit crops were made in accordance with the customary application schedule and the crops analyzed for pesticide residues at harvest time. BHC appeared to be most readily taken up, followed by endrin while DDT and aldrin were less readily taken up. Previous studies had indicated that aldrin is oxidized to dieldrin in plant tissues after uptake; however, on the basis of the present studies, it is proposed that aldrin is first oxidized to dieldrin in the soil, then the dieldrin taken up by the plant. Oil-bearing plants had higher pesticide residues than the other plants tested and contained large quantities of residues in subterranean stems.

73-0571. Takanuma, S.; Wada, T. (Nagano Prefect. Agr. Exp. Station, Nagano, Japan). **[Results of investigation of organochlorine pesticide residues in soils where vegetables are grown.]** *Kanto Tosan Byogaichu Kenkyukai Nenpo (Proc. Kanto-Tosan Plant Prot. Soc.) 19: 124-125; 1972.* (Japanese)

Results of investigations in 1970 of organochlorine pesticide residues in 32 soil specimens in vegetable fields in Nagano Prefecture are reported. The sampling and analysis of the soil specimens were carried out with standard methods and GC. Four vegetable fields where rice was grown previously for 4-5 years and four regular vegetable fields were used. The smaller the amount of application the smaller the residue. In the converted fields the residue was relatively small despite the relatively large amounts of applied BHC. Endrin residues did not parallel the amount of the application. The correlation between the residues and pH and organic matters in the soil were higher for δ-BHC and dieldrin. The smaller residue of DDT was found in the soil with a high pH. The correlation between total carbon and the residues was high for α- and γ- BHC. There was also a high correlation between humus and γ-BHC and humic acid and γ- and α-BHC.

73-0572. Akagi, H.; Sakagami, Y. (Dept. of Pub. Health Pharmaceutics, Inst. of Pub. Health, Tokyo, Japan). **[On degradation of organotin compounds by ultra violet ray.]** *Koshu Eiseiin Kenkyu Hokoku (Bull. Inst. Pub. Health) 20(1): 1-4; 1971.* (8 references) (Japanese)

Organotin compounds are used as agricultural fungicides as well as stabilizers for plastics. Thin-layer chromatography was used to investigate the degradation of organotin compounds during exposure to ultraviolet radiation. Aliquots were taken from solutions of tin compounds (including triphenyltin chloride and tributyltin chloride) after specified irridiation times and spotted onto silica gel layers containing 10% ammonium sulfate. The chromatograms were developed with various solvent systems and the degradation products detected by spraying with dithizone or pyrocatechol violet. Triphenyltin chloride was converted into a mixture of triphenyltin, diphenyltin, monophenyltin, and inorganic tin compounds within six hours; degradation of all triphenyltin to tin chloride required ca. 325 hr of irradiation. Inorganic tin compounds comprised the ultimate degradation products in all cases.

73-0573. Brueggemann, J. (Author address not given). **Wild als Giftindikator. [Wildlife as a poison indicator.]** *Kosmos 68(11): 472-473; 1972.* (German)

The combined residue content of 12 organochlorine insecticides was determined in 100 liver and 90 fat samples from wild animals. The total residue content in rabbit liver and fat was 0.03 ppm and 2 ppm, respectively. The corresponding residue contents in fox liver were more than 10 times higher; that in rapacious birds' eggs was 100 to 1,000 times higher. The DDT residue contents were in an order of magnitude of 0.002 ppm, except in fox and in rapacious birds' eggs, in which considerably higher values were determined.

73-0574. Annett, C. S.; Fadow, M. P.; D'Itri, F. M.; Stephenson, M. E. (Michigan State Univ., East Lansing, MI). **Mercury pollution and Lake Erie fishes.** *Mich. Acad. 4(3): 325-337; 1972.* (26 references)

Most mercury compounds released into the environment can be directly or indirectly transformed into highly toxic mono- and dimethylmercury. Methylmercury accumulation in aquatic organisms is probably a function of the metabolic rate of the individual fish, differences in the selection of food as the fish matures, and the fish's surface area. The group of fish, mostly predators, accumulating the highest quantities of mercury includes muskellunge, pike, bass, and walleye. Sedimentation can remove mercury from water. According to a Federal Water Quality Administration survey in the spring of 1970, the sediment in the Raisin River area contained less than 1.8 ppm total mercury on a dry weight basis. In the study reported, total mercury in sediments taken from the extreme western basin of Lake Erie near the Raisin River in the fall of 1970 was evenly distributed and ranged from 0.19 to 0.53 ppm on a dry weight basis. Surface water samples were always less than 0.1 ppb except for values of 0.2 and 0.7 ppb found at stations which might have been points of mercury discharge. Overall concentrations in fish were lower than those for the same species taken from Lake St. Clair. The mean total mercury levels were 0.49 ppm in perch, 0.28 ppm in goldfish, and 0.19 ppm in carp.

73-0575. Miyabe, M.; Tsubouchi, H.; Sakabe, Y. (Dept. of Foods, Nagoya Municipal Pub. Health Lab., Nagoya, Japan). **[Studies on pesticide residues in foods. II. Residues of BHC in milk.]** *Nagoya-shi Eisei Kenkyusho-ho (Rep. Nagoya Munic. Pub. Health Lab.) 18: 37-41; 1971.* (11 references) (Japanese)

Samples of milk from a milk treatment factory were analyzed monthly from December 1969 to March 1972. The determination of isomers of BHC was carried out with GC. The highest value of total BHC in 1969 was 1.7 ppm in whole milk, 56 ppm calculated on fat basis, and very high as compared to the BHC content in other foods. Although the values fluctuated, they gradually decreased with time. The total average value stabilized at around 0.09 ppm in whole milk in September 1970, and further decreased to about 0.04 ppm. The lowest value recorded was 0.02-0.03 ppm in the same month when the pollution had been relatively high. Of the four major isomers β-BHC decreased from 1969 to 1971 to an average value of 0.020-0.025 ppm, the highest value being stabilized about 0.04 ppm. Alpha-BHC to total BHC was relatively high when the pollution was high and decreased slowly. Beta-BHC increased and stabilized at 60-70% of the total BHC. Gamma-BHC (lindane) remained steady at 2-5% of total BHC then increased slowly to 20%. Delta-BHC stabilized at a value between 2-5%. The BHC content in milk decreased as compared to that of before 1970, 0.02 ppm of β-BHC and 0.03% of total BHC; the highest value in each month shows considerable fluctuation, suggesting the necessity of continued monitoring.

73-0576. Koelle, W.; Ruf, H.; Stieglitz, L. (Inst. fuer Radiochem. Kernforschungs-zentrum, Karlsruhe, Germany). **Die Belastung des Rheins mit organischen Schadstoffen. [The pollution of the Rhine by organic substances.]** *Naturwissenschaften 59(7): 299-305; 1972.* (15 references) (German)

The overall pollution level of a body of water must be assessed from several viewpoints. One is the total quantity of substances of various classes present; another is the quantity of individual substances present considered particularly undesirable. Overall water quality analyses include determinations of the biochemical oxygen demand (BOD), UV extinction, and organic carbon content (dissolved and suspended); radiocarbon dating for distinguishing between fossil (petroleum) and non-fossil carbon contamination; and organic chlorine

analysis. Specific substances which must be sought include pesticides (particularly organochlorines, but also organophosphates), aromatic hydrocarbons, and surfactants. Gas chromatographic analysis of fish specimens collected along the Rhine from Lake Constance to Dusseldorf revealed that contamination with aliphatic and aromatic, chlorinated and non-chlorinated hydrocarbons increased progressively downstream. The only pesticide identified in the chromatograms was p,p'-DDE; quantitation was not attempted.

73-0577. Itzuka, H.; Kanazawa, J.; Miyahara, K. (Nat. Inst. of Agr. Sci., Nishigahara, Tokyo, Japan). **[Studies on residues of BHC applied in paddy fields.]** *Nippon Oyo Dobutsu Konchu Gakkai-shi (Jap. J. Appl. Entomol.) 16(3): 139-147; 1972.* (13 references) (Japanese)

BHC residues in soil, hulled rice, and rice straw were examined in samples collected from paddy fields at Saga and Kagawa Agricultural Experiment Stations. BHC residues in hulled rice were mathematically related to both the spray history of the fields in question and the BHC residues in surrounding fields. One year after the application of BHC was prohibited in Japan, the BHC residue content in hulled rice had dropped by one-third to one-fifth. A significant mathematical correlation was found between the log of the residual BHC content in soil and that in hulled rice. Uptake of BHC in rice plants is apparently inhibited by high organic matter contents in the soil. The majority of the BHC, especially the β-isomer, was located close to the soil surface. The relative contents of the BHC isomers in hulled rice, straws, and soils did not correlate with the contents in technical products; the β/γ (lindane) ratio in the plants was increased by a factor of 10, and the δ/γ ratio by a factor of 3 to 5 compared to the technical products.

73-0578. Belisle, A. A.; Reichel, W. L.; Locke, L. N.; Lamont, T. G.; Mulhern, B. M.; Prouty, R. M.; DeWolf, R. B.; Cromartie, E. (Bureau of Sport Fisheries and Wildl., Patuxent Wildl. Res. Cent., Laurel, MD 20810). **Residues of organochlorine pesticides, polychlorinated biphenyls, and mercury and autopsy data for bald eagles, 1969 and 1970.** *Pestic. Monit. J. 6(3): 133-138; 1972.* (6 references)

Thirty-nine bald eagles found sick or dead in 13 states during 1969 and 1970 were analyzed for pesticide residues. Residues of DDE, dieldrin, polychlorinated biphenyls (PCBs), and mercury were detected in all bald eagle carcasses; DDD (TDE) residues were detected in 38; DDT, heptachlor epoxide, and dichlorobenzophenone (DCBP) were detected less frequently. Six eagles contained possible lethal levels of dieldrin in the brain, and one contained a lethal concentration of DDD (385 ppm) in the brain together with 235 ppm of PCBs. Autopsy revealed that 18 bald eagles were illegally shot; other causes of death were impact injuries, electrocution, emaciation, and infectious diseases. (Author abstract by permission)

73-0579. Giam, C. S.; Hanks, A. R.; Richardson, R. L.; Sackett, W. M.; Wong, M. K. (Dept. of Chem., Texas A&M Univ., College Station, TX 77843). **DDT, DDE, and polychlorinated biphenyls in biota from the Gulf of Mexico and Caribbean Sea–1971.** *Pestic. Monit. J. 6(3): 139-143; 1972.* (9 references)

Residue levels of DDT, DDE, and PCBs were determined in various species of fish, shrimp, crabs, and other biota from the Gulf of Mexico and Caribbean Sea. Samples were collected from the Gulf during two Gulf-wide cruises in May and October 1971 and from part of the Caribbean Sea during the October cruise. DDT, DDE, and PCBs were found widely distributed in all biota; however, samples from coastal areas generally had higher levels than samples from the open water. (Author abstract by permission)

73-0580. Henderson, C.; Inglis, A.; Johnson, W. L. (Div. of Fishery Services, Bureau of Sport Fisheries and Wildl., U.S. Dept. of the Interior, Washington, DC 20240). **Mercury residues in fish, 1969-1970–National Pesticide Monitoring Program.** *Pestic. Monit. J. 6(3): 144-159; 1972.* (8 references)

As part of the fish monitoring program conducted by the Bureau of Sport Fisheries and Wildlife since 1967, composite fish samples collected during the fall of 1969 and 1970 were analyzed for mercury. Three composite samples, each of a different species and consisting of 3-5 adult fish, were collected at each of 50 monitoring stations in 1969; similarly, three composite samples, and in most cases a replicate sample of one of the species, were collected at each of 100 stations in 1970. Stations were located on major rivers and lakes throughout the United States. Total mercury residues equal to or exceeding the sensitivity level of 0.05 ppm were found in 129 of the 145 samples in 1969 and 373 of the 393 samples in 1970. Values ranged from <0.05 to 1.25 ppm in 1969 samples and from <0.05 to 1.80 ppm in 1970 samples. Analyses by two different laboratories of 40 selected samples from the 1970 collection gave comparable results. Analyses of 24 selected 1970 samples indicated that 90% or more of the mercury in fish was in the form of methyl mercury. (Author abstract by permission)

73-0581. Marganian, V. M.; Wall, Jr., W. J. (Dept. of Chem., Bridgewater State College, Bridgewater, MA 03324). **Dursban and diazinon residues in biota following treatment of intertidal plots on Cape Cod–1967-1969.** *Pestic. Monit. J. 6(3): 160-165; 1972.* (9 references)

The effects of the organophosphorus pesticides, Dursban (chlorpyrifos) and diazinon, on intertidal biota

were studied over a period of three years, 1967-69. One percent granular Dursban applied manually at an optimum concentration of 0.05 lb/acre controlled *Culicoides* larvae effectively with no noticeable harm to fiddler crabs or other organisms. Residues recovered ranged from trace amounts to 2.30 ppm in white oligochaete, 2.58 ppm in ribbed mussel, 4.62 ppm in fiddler crab, 14.0 ppm in horsefly, and 15.7 ppm in marsh snail. Two percent granular diazinon applied manually at 0.20 lb/acre controlled *Culicoides* effectively, but killed small sand organisms. In general, concentrations of diazinon residues recovered were higher than those for Dursban in the same organisms reported above. This report describes the sampling and gas chromatographic analytical procedures employed in this work, discusses data collected on residues in organisms at various periods after treatment, and gives persistence periods for these pesticides in substrates of intertidal sand, salt marsh sod, salt marsh mud, and sea water. (Author abstract by permission)

73-0582. Bradshaw, J. S.; Loveridge, E. L.; Rippee, K. P.; Peterson, J. L.; White, D. A.; Barton, J. R.; Fuhriman, D. K. (Chem. Dept., Brigham Young Univ., Provo, UT 84601). **Seasonal variations in residues of chlorinated hydrocarbon pesticides in the water of the Utah Lake drainage system–1970 and 1971.** *Pestic. Monit. J. 6(3): 166-170; 1972.* (13 references)

Definite surges of pesticides (1 ppb or more) enter Utah Lake three times per year–early spring, late spring, and fall, generally corresponding to the application times of pesticides by farmers in the area. The pesticides involved were mainly aldrin and BHC in the early spring; heptachlor (plus heptachlor epoxide) and methoxychlor in the late spring; and aldrin, heptachlor, and methoxychlor in the late fall. The fish samples collected from Utah Lake contained only small amounts of pesticides, the highest level being 956 ppb DDE. (Author abstract by permission)

73-0583. Feltz, H. R.; Culbertson, J. K. (U. S. Geological Survey, Arlington, VA 22209). **Sampling procedures and problems in determining pesticide residues in the hydrologic environment.** *Pestic. Monit. J. 6(3): 171-178; 1972.* (18 references)

Diligent use of standardized sampling and analytical techniques is essential to meaningful assessments of the occurrence, distribution, and fate of pesticide residues in the hydrologic environment. The validity of analytical data and subsequent interpretations are interdependent and limited to the confidence level of adequate, representative sampling of various components. Equally important are appropriate sample-preservation practices and procedures for sample preparation and cleanup and identification, measurement, and confirmation of residues. Analytical schedules should include pesticides listed in the Revised Chemicals Monitoring Guide for the National Pesticide Monitoring Program and should be responsive to special interests. Samplers are available for collecting acceptable water, fluvial material, and bottom-material samples in about 75% of the river miles and in lakes and estuaries throughout the United States; however, more experience is needed in sampling hydrosols and low density deposits at the active water-sediment interface. (Author abstract by permission)

73-0584. Herzel, F. (Inst. fuer Wasser-, Boden-, und Lufthygiene des Bundesgesundheitsamtes, Berlin, Germany). **Organochlorine insecticides in surface waters in Germany–1970 and 1971.** *Pestic. Monit. J. 6(3): 179-187; 1972.* (36 references)

As part of a series of studies initiated in 1969 to determine the organochlorine insecticide content of major waters in the Federal Republic of Germany, unfiltered water and suspended solids were analyzed from approximately 25 sites sampled in May 1971, and unfiltered water was analyzed from seven sites sampled monthly from April 1970–June 1971. As in former studies (June and October 1969, April and September 1970), the insecticide concentrations found in waters and suspended solids were almost exclusively in the ppt range (ng/liter). The compounds found most frequently were γ-BHC (lindane) and α-BHC; α- and β-endosulfan were detected in the Main, Regnitz, and Rhine Rivers. DDT and particularly its metabolites DDD (TDE) and DDE were found infrequently except in samples from the Berlin Teltowkanal. Findings of heptachlor, heptachlor epoxide, dieldrin, and parathion (the only organophosphorus insecticide included in the study) were rare. (Author abstract by permission)

73-0585. Lenon, H.; Curry, L.; Miller, A.; Patulski, D. (Dept. of Biol., Central Michigan Univ., Mt. Pleasant, MI 48858). **Insecticide residues in water and sediment from cisterns on the U.S. and British Virgin Islands–1970.** *Pestic. Monit. J. 6(3): 188-193; 1972.* (6 references)

In the Virgin Islands the potential exists for pesticide contamination of water cisterns which supply and store all water used by local populations; cistern water and sediment samples on four islands were analyzed for pesticide residues in 1970 by gas-liquid chromatography. In the past, chlorinated hydrocarbon pesticides were used quite extensively on the Islands, however, malathion is more commonly used today. Evidence of an unknown malathion metabolite was found in all 49 water samples analyzed, whereas malathion was found in only two (0.01 and 0.14 ppb). DDT, its metabolites, and dieldrin were not commonly found in the water samples except those from St. John where dieldrin was detected in approximately 50% of the samples (average concentration–0.19 ppb). Sediment samples from cisterns, in general, contained much higher concentrations of pesticides than water, with DDT and its metabolites occurring most frequently. In many of these sediment samples, the residue levels

were high enough to cause concern. As a result it is strongly recommended that cisterns be cleaned frequently to remove sediment. (Author abstract by permission)

73-0586. Wiersma, G. B.; Tai, H.; Sand, P. F. (Pesticides Regulation Div., Office of Pesticide Programs, Environmental Protection Agency, Washington, DC 20460). **Pesticide residue levels in soil, FY 1969–National Soils Monitoring Program.** *Pestic. Monit. J. 6(3): 194-228; 1972.* (5 references)

This report is a summary of the FY 1969 results of the National Soils Monitoring Program, an integral part of the National Pesticide Monitoring Program (NPMP). Pesticide residues in cropland soil for 43 states and noncropland soil for 11 states are reported. Tables for each state give the number of samples collected, arithmetic means, and ranges of residue levels detected, and the percent of sites with detectable residues. In addition, for selected pesticides and various states and state groupings, a frequency distribution of pesticide residues was determined. Use records for FY 1969 are given by the pesticides used, the percent of sites treated, the average application rates, and the average amounts applied per site. Comparisons are made between residue levels in different land-use areas. (Author abstract by permission)

73-0587. Check, R. M.; Canario, Jr., M. T. (Div. of Laboratories, Rhode Island Dept. of Health, Providence, RI 02903). **Residues of chlorinated hydrocarbon pesticides in the northern quahog (hard-shell clam), *Mercenaria mercenaria*–1968 and 1969.** *Pestic. Monit. J. 6(3): 229-230; 1972.* (3 references)

Samples of the northern quahog (hard-shell clam), *Mercenaria mercenaria*, were collected monthly, when possible, from September 1968 to September 1969 at five locations in Narragansett Bay, Rhode Island, and one location in nearby Mount Hope Bay. All 56 composite samples contained dieldrin at an average level of 0.040 ppm; *p,p'*-DDD (TDE) was present in three samples at an average level of 0.026 ppm. Quahogs from upper reaches of Narragansett Bay contained higher levels of residues than samples from lower Bay areas. (Author abstract by permission)

73-0588. Inch, T. D.; Ley, R. V.; Utley, D. (Chemical Defense Establishment, Porton Down, Salisbury, Wilts., England). **The mobility of some organophosphorus sheep dip insecticides in soil.** *Pestic. Sci. 3(3): 243-253; 1972.* (18 references)

The technique of soil thin-layer chromatography has been adapted to permit the use of repeated or prolonged irrigation procedures so that the mobility characteristics of relatively non-mobile insecticides may be studied. The procedure has been used to study the relative movement of organophosphorus insecticides such as chlorfenvinphos, Trithion (carbofenothion), Delnav (dioxathion), and dichlofenthion which are widely used as sheep dips. All pesticides tested had very low mobility; the slight migration of radiolabeled compounds was apparently due to washout of fine soil particles containing adsorbed pesticides. These results are in agreement with field experiments which showed minimal leaching of chlorfenvinphos in five months (total rainfall, 30 cm). Soil thin-layer chromatography might be useful in studying breakdown of pesticides in soil. Since the intact insecticides are generally more firmly adsorbed by soil than are the breakdown products, the eluents from chromatographic experiments could be analyzed for metabolite content. (Author abstract by permission, supplemented)

73-0589. Beynon, K. I.; Wright, A. N. (Woodstock Agr. Res. Cent., Sittingbourne, Kent, England). **The breakdown of [^{14}C] monocrotophos insecticide on maize, cabbage, and apple.** *Pestic. Sci. 3(3): 277-292; 1972.* (4 references)

The breakdown of ^{14}C-labeled monocrotophos (dimethyl 3-hydroxy-*N*-methyl-*cis*-crotonamide phosphate) on the foliage of maize, cabbage, and apple, and on apple fruit has been studied. The breakdown products were mainly hydrophilic compounds such as *O*-desmethyl-monocrotophos acid and probably dimethyl phosphate. Other breakdown products were *N*-methylacetoacetamide, the *N*-hydroxymethyl derivative (free and conjugated), the amide, and 3-hydroxy-*N*-methylbutyramide. In maize and apple the *N*-hydroxymethyl derivative was conjugated with sugars other than β-D-glucose, and a polar component, possibly a conjugate, was present in cabbage, but this may not have been derived from the *N*-hydroxymethyl derivative. (Author abstract by permission)

73-0590. Beynon, K. I.; Stoydin, G.; Wright, A. N. (Shell Res. Ltd., Woodstock Agr. Res. Cent., Sittingbourne, Kent, England). **The breakdown of the triazine herbicide cyanazine in soils and maize.** *Pestic. Sci. 3(3): 293-305; 1972.* (15 references)

Radioisotope techniques have been used to study the breakdown products that are formed from the herbicide cyanazine (Bladex), 2-chloro-4-(1-cyano-1-methylethyl-amino)-6-ethylamino-1,3,5-triazine, in soils and in maize grown in the soils under indoor conditions. In soils of different types cyanazine broke down mainly by conversion of the nitrile group to amide and then to an acid followed by hydrolysis of the ring chlorine to hydroxyl. Dealkylation reactions occurred to only a limited extent in soils. In maize plants grown in treated soils the hydrolysis products, the amide and the hydroxy acid were detected as well as appreciable quantities of products formed from these by loss of the *N*-ethyl group. In plants the hydroxy acids were present in the free form, and there was also evidence for conjugates

which were not identified but could be converted to these hydroxy acids on treatment with acids. In these indoor studies the major residues appear to be the hydroxy acid in soils and the hydroxy acid and its dealkylated analogue in plants grown in treated soils. These compounds are not herbicides and are of a low order of toxicity to mammals. (Author abstract by permission)

73-0591. Visweswariah, K.; Jayaram, M. (Central Food Technological Res. Inst., Mysore, India). **The effect of processing in lowering the BHC residue in green leafy vegetables.** *Pestic. Sci. 3(3): 345-349; 1972.* (12 references)

Investigations conducted on certain varieties of green leafy vegetables revealed very high BHC residues. The significance of certain common processing techniques in reducing the pesticide residue level on the raw material was investigated. Cooking the material in water for five minutes and immersion of the sample in 1% acetic acid solution and subsequent cooking reduced the level of pesticide residue within the permissible limit. (Author abstract by permission)

73-0592. Pitblado, R. E.; Edgington, L. V. (Dept. of Environ. Biol., Univ. of Guelph, Guelph, Ontario, Canada). **Movement of benomyl in field soils as influenced by acid surfactants.** *Phytopathology 62(5): 513-516; 1972.* (12 references)

Several surfactants increased the water solubility of methyl 2-benzimidazolecarbamate (MBC), the fungicidal breakdown product of benomyl, and its penetration in two types of field soil. A 1% concentration of the acidic surfactant GAFAC RA-600 (General Aniline and Film Corp.) which had a pH of 2.7 or of Tween 20 adjusted to pH 2.7 enhanced the solubility of MBC about 100 fold while phosphoric acid alone increased the solubility about 75 fold. The movement of benomyl, or more precisely its breakdown product, was greatest with the addition of the GAFAC surfactants or acid plus Tween 20, but was also enhanced by Tween 20 alone. The buffering capacity of the soil caused precipitation of acidified MBC. In the dry soil MBC did not penetrate so far, but the relative differences in downward movement between mixtures remained constant.

73-0593. Hughes, J. T. and Woodwell, G. M.; Craig, P. P.; Johnson, H. A. (Chem. Div., Dept. of Sci. and Ind. Res., Private Bag, Petone, New Zealand and Brookhaven Nat. Lab., Upton, NY 11973). **DDT in rainfall.** *Science 178(4060): 450; 1972.* (1 reference)

Several corrections are made to the paper by Woodwell, Craig, and Johnson entitled "What Happens to DDT" (see Abstract 72-0246). Results quoted for the amount of DDT found in rain in England were given in parts per 10^{12}, not in parts per million. A typographical error in the formula in the section entitled "A Model of DDT Circulation in the Biosphere" showed *Ni* in the second summation instead of *Nj*.

73-0594. Uyeta, M.; Taue, S.; Nishimoto, T. (Kochi Prefect. Pub. Health Lab., 5 Marunouchi, Kochi, Japan). **Study of pesticide residues in food (V). Residues of BHC isomers and other organochlorine pesticides in fatty foods of Japan.** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 11(4): 256-263; 1970.* (18 references)

Persistent organochlorines were determined in 56 samples of milk, cheese, and butter and in 40 samples of meats and eggs produced in Japan. α, β, γ and δ-BHC, *p,p'*-DDE, *p,p'*-DDT, and *p,p'*-DDD (TDE), dieldrin, endrin, and heptachlor epoxide were found, aldrin and heptachlor were not. Levels in whole milk ranged from 0.059 to 0.356 ppm for total BHC, 0.006 to 0.037 ppm for total DDT, and 0.002 to 0.010 ppm for dieldrin. Levels on a fat basis in meats ranged from 0.50 to 13.68 ppm for total BHC, 0.29 to 0.56 ppm for total DDT, and 0.01 to 0.25 ppm for dieldrin. β-BHC, which is reported to have lower acute toxicity but higher chronic toxicity than the other isomers, made up about 70% of the total BHC residues. BHC levels were much higher than those in other countries and indicate a need to curtail the large-scale use of BHC in Japan.

73-0595. Nishimoto, T.; Uyeta, M.; Taue, S.; Chikazawa, K. (Kochi Prefect. Pub. Health Lab., Kochi, Japan). **[Study of pesticide residues in food (VI). Residues of drin-pesticides and other organochlorine pesticides in vegetables.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 12(1): 56-61; 1971.* (18 references) (Japanese)

Organochlorine pesticide contents were determined in 132 samples of nine kinds of vegetables purchased at markets in Tokyo, Osaka, and Kochi in June-August, 1970. Residues greater than 0.03 ppm aldrin + dieldrin and/or 0.01 ppm endrin were found in 21 of 35 cucumber samples, 9 of 17 carrot samples, 8 of 18 potato samples, 8 of 15 sweet potato samples, and one sample each of cabbage, chinese cabbage, and lettuce. Total BHC residues of more than 0.5 ppm were found only in one carrot and one cabbage sample. In the soil of fields formerly used for growing tobacco (sprayed with aldrin), then planted with cucumbers, aldrin/dieldrin declined from 0.6 ppm in the first year after the last aldrin application to 0.3 ppm in the second year, 0.1 ppm in the third year, and 0.03 ppm in the fourth year. Aldrin/dieldrin residues in cucumbers grown in these fields in the successive years were 0.07, 0.03, 0.02, and 0.003 ppm.

73-0596. Study Group Min. Health-Welfare for Organochlorine Pestic. Residues in Mothers' Milk. (National Inst. of Hygienic Sci., Setagaya, Tokyo, Japan). **[Studies**

on the analysis of pesticide residues in foods. (VII) Organochlorine pesticide residues in mothers' milk.] *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 13(5): 422-437; 1972.* (9 references) (Japanese)

The residue content of α-, β-, γ-, and δ-BHC, *p,p'*-DDT, *p,p'*-DDE, *p,p'*-DDD (TDE), and polychlorocyclodienes were analyzed in 454 specimens of human milk from 24 prefectures in Japan. The specimens were taken from 20 nursing mothers more than one month post partum. The method of extracting fats from the milk specimens and purification are described. Dietary habits were also investigated. Beta-BHC was detected in almost all the specimens in amounts ranging from 0.0330 to 0.44 ppm on a whole milk basis. The content was higher in western districts than in eastern districts and higher in urban women rather than in rural women in the former. Eighty-four percent of all the specimens contained less than 0.2 ppm of β-BHC (the temporary tolerance in cows' milk). The percentage of detection of γ-BHC (lindane) was 96 although the content was low, from 0.000 to 0.10. The percentage of detection of α- and δ-BHC was 99 and 45, respectively. The average contents of isomers of BHC in all 454 specimens were 0.0003, 0.12, 0.0003, and 0.001 ppm respectively for α-, c-, γ-, and δ-BHC; the ratio was 2.5:95:2.0:0.5, suggesting the bio-concentration of β-isomer in humans as in cows. Total DDT was detected in all the specimens. Although *p,p'*-DDE was detected in almost all the specimens, no *p,p'*-DDD (TDE) was detected. Seventy-nine percent of the specimens contained dieldrin; however, the average concentration was 0.004 ppm (temporary tolerance is 0.005 in cows' milk). Only two cases of aldrin were found and no cases of endrin. Some relationships were found between the content of β-BHC, *p,p'*-DDT, and dieldrin in the mothers' milk and the amount of beef and milk in the diet. However, these were only significant with β-BHC. No such relationship was found with drinking water, butter, vegetables, and cigarette smoking.

73-0597. Study Group Min. Health-Welfare for Organochlorine Pestic. Residues in Mothers' Milk. (National Inst. of Hyg. Sci., Setagaya, Tokyo, Japan). **[Studies on the analysis of pesticide residues in foods. (VIII) Organochlorine pesticide residues in blood during lactation.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 13(5): 438-450; 1972.* (7 references) (Japanese)

Women participating in the residues in human milk project were also tested for residues in blood. Blood was collected at the same time as milk sampling and immersed into 0.1 ml heparin solution to avoid coagulation. Six ml of the blood was used for the determination of organochlorine pesticide residues, the remainder being chilled and kept for examining liver function. The pesticide residues were extracted by the method of Dale (*J. Ass. Offic. Anal. Chem.* 53: 1287; 1970) and after cleanup, β-BHC, γ-BHC, *p,p'*-DDT and dieldrin were detected by GC with average percentage recovery of 71.6, 74.5, 76.6, and 70.0, respectively. Levels of β-BHC were relatively lower in Hokkaido and three northeastern prefectures than in others; no tendency of higher pollution in western districts such as found in mothers' milk was noticed. Especially high β-BHC levels were found in urban women in Osaka (0.050 ppm), in rural and urban women in Aichi (0.029 and 0.022), in urban women in Kyoto (0.030), and in urban women in Hiroshima (0.032). The γ-BHC (lindane) was highest in Akita (0.008 in rural and 0.011 in urban women), Kyoto (0.017 and 0.010 ppm), Fukushima, Aichi, Osaka, and Hyogo. Of the total 410 specimens 52% contained γ-BHC less than traces. The percentage of the specimens containing larger than 0.005 ppm of total BHC was 88 on the average. *p,p'*-DDT was found in all the blood specimens in nearly the same level (50% of the specimens contained less than 0.001 ppm). Total DDT was found in all the specimens at nearly the same level except in a few prefectures such as Hiroshima (0.078), Kyoto (0.049), and Osaka (0.049 ppm). *p,p'*-DDE was detected in all the specimens; no *p,p'*-DDD (TDE) was detected. Dieldrin was detected in all the specimens at a level less than 0.003 ppm. No aldrin nor endrin were detected.

73-0598. Yamada, T.; Kato, K.; Watanabe, S.; Nakaoka, M.; Wada, H. (Kanagawa-Prefect. Pub. Health Res. Lab.). **[A trend of pesticide residues in milk of individual cows.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 13(5): 479; 1972.* (Japanese)

Raw milk specimens were taken from 18 cows four times in each of four series and analyzed for α-, β- and γ-BHC, *p,p'*-DDT, *p,p'*-DDE, aldrin, and endrin. The analysis was carried out with GC with ECD with a column packed with OV-17, DEGS plus phosphoric acid. The cows' feed was also examined. The range of total BHC (α+β+γ) was 0.0078-0.0878 ppm, that of total DDT (*p,p'*-DDT+*p,p'*-DDE) was 0.0004-0.0193 ppm, and that of dieldrin was 0.0003-0.0031 ppm. No cases exceeded the temporary tolerance in cows' milk decided by the Ministry of Health and Public Welfare in Japan. Neither aldrin nor endrin were detected. The average residue of total BHC in the specimen collected in May 1972 was one third of that in August 1971. With total DDT, the corresponding ratio was one fourth, the same trend was also found in dieldrin. Residues were present in the feed, especially in rice straw.

73-0599. Place, M.; Dutremee, C. (Inst. Super. d'Agr. de Lille, Lille, France). **Les residus de pesticides dans le lait. Resume du projet de fin d'etudes a l'I.S.A. [Pesticide residues in milk. Summary of an I.S.A. research project.]** *Tech. Lait. 27(34): 27-31; 1972.* (French)

General problems involved in the elimination of organochlorine pesticide residues in milk and milk products and experiments on the elimination of artifically introduced pesticides in cows are described. Milk

samples investigated contained 0.95-1.3 ppm of total BHC and 0.45 ppm of heptachlor epoxide. Despite the 1971 ban on the use of certain organochlorine pesticides for disinsection of stables, the contamination level of milk has remained fairly high although the gamma-BHC (lindane) contents are usually small. Experiments with ectodermal application of organochlorine pesticides or admixture to feed show fairly rapid elimination of the BHC isomers in the milk and relatively slow rate of elimination of heptachlor epoxide and dieldrin.

73-0600. Marking, L. L. (Fish Control Lab., P.O. Box 862, La Crosse, WI 54601). **Salicylanilide I, an effective non-persistent candidate piscicide.** *Trans. Amer. Fish. Soc. 101(3): 526-533; 1972.* (16 references)

Salicylanilide I, a possible piscicide, was found to detoxify in water, the rate being slowest in cold, neutral water. Results of laboratory bioassays, in which 96-hr LC50s of the compound for 20 specimens of fish ranged from 0.3 to 8.6 ppb, suggested that lethal concentrations detoxify at 12 to 17°C in less than four days. In outdoor tests salicylanilide I was less toxic, probably because of inactivation by chemical, biological, or physical factors such as light and temperature. In such tests with rainbow trout, temperatures went down to freezing and the chemical remained toxic for some time.

73-0601. Acker, L.; Schülte, E. (Inst. fuer Lebensmittelchemie, Univ. Muenster, Muenster, Germany). **Chlorierte Kohlenwasserstoffe im menschlichen Koerper. [Organochlorine compounds in the human body.]** *Umschau 71(23): 848; 1971.* (3 references) (German)

DDT, beta-BHC, dieldrin, PCB, and hexachlorobenzene residue contents were determined in human fat and in human milk fat. Human adipose tissue samples from the Muenster region contained, on an average, 3.8 ppm of DDT plus DDE, 0.5 ppm of beta-BHC, and 0.2 ppm of dieldrin. The PCB residue concentrations in human fat and in human milk fat were 5.7 ppm and 3.5 ppm, respectively. Hexachlorobenzene residue contents of 6.3 ppm in human fat and of 5.3 ppm in human milk fat were determined.

73-0602. Seehofer, F.; Nesemann, E. (BAT Cigaretten-Fabrik GmbH, Hamburg, Germany). **Pestizidrueckstaende im Tabak. [Pesticide residues in tobacco.]** *Umschau 72(20): 661; 1972.* (1 reference) (German)

The problem of pesticide residues and their reduction in tobacco is outlined. Drying, curing, and processing result in elimination of up to 60% of the organochlorine insecticide and dithiocarbamate residues and in up to 99% of the organophosphorus compounds and carbamates. Smoking as well as the blending of tobaccos of various locations further reduce the pesticide residue contents. Modern analytical methods (gas chromatography, electrometric and spectrometric procedures) allow the detection of pesticide residues of 1 ng in tobacco.

73-0603. Dimitrov, S.; Naydenov, N.; Tamarov, B.; Vuleva, R.; Sapundzhieva, L. (Inst. Biol. and Pathol. of Reprod. and Noninfectious Diseases, Sofia, Bulgaria) **Izsledvane na trevni furazhi za ostatuchni kolichestva ot khlororganichni i fosfororganichni insektisidi. [Investigation of grass forages for residual amounts of organochlorine and organophosphate insecticides.]** *Vet. Med. Nauki 8(8): 77-82; 1971.* (20 references) (Bulgarian)

Organochlorine and organophosphate residues were studied in grass forage samples, collected from different regions in Bulgaria; in wheat prior to harvesting of the crop and feeding of the straw; and in alfalfa meadow and orchard hay after mowing and during cattle feeding. Wheat straw had no parathion, fostion (prothoate), or dipterex (trichlorfon) residues while fed to livestock. Their levels were found to decrease to 0.1 mg/kg fodder within 30 days after application. Blood cholinesterase activities determined in 12 cows fed the above were normal. Hay samples from meadow and orchard alfalfa, previously subjected to organochlorine insecticide treatment, contained considerable DDT residues during a six month feeding period. Some of these samples contained traces of aldrin also. The need for thorough studies in establishing a safe hay decontamination quarantine is emphasized.

73-0604. Keymer, I. F.; Smith, G. R.; Roberts, T. A.; Heaney, S. I.; Hibberd, D. J. (Zool. Soc. of London, Regent's Park, London, England). **Botulism as a factor in waterfowl mortality at St. James's Park, London.** *Vet. Rec. 90(5): 111-114; 1972.* (11 references)

During a period of unusually warm weather in London, in June-November 1969, approximately 40% of the 1000 waterfowl on the St. James's Park lake died. The lake and the bird carcasses were examined in an attempt to determine the cause of the mortality. A heavy blue-green algal bloom was present in the lake water during the period, but similar blooms had also been seen in years when no bird deaths occurred. The chief symptom in sick birds was progressive weakness. Botulinus toxin was detected at concentrations sufficient to cause death in five of the 16 carcasses examined. The carcasses of two affected mallards contained traces of DDE (1.0 and 0.8 ppm). One bird contained 0.2 ppm dieldrin. No TDE, DDT, BHC, or other organochlorines were found. These pesticide contamination levels are comparatively low. No significant macroscopic lesions were observed. Histological examination of livers and kidneys of dead birds revealed generalized congestion and slight fatty infiltration. Botulism was considered the most likely cause of death.

73-0605. Wolter, D. (Hygiene-Institut des Bezirkes Schwerin, Schwerin, DDR). **DDT in Oberflaechengewaessern. [DDT in surface waters.]** *Z. Gesamte Hyg. 18(4): 247-248; 1972.* (9 references) (German)

Results of DDT residue determinations in surface water and in drinking water and recommendations for

the restriction of DDT use are presented. A maximal DDT residue content of 0.5 mg/l. was measured in surface water immediately following aircraft distribution of DDT over colza fields. The residue content was persistently at a level of 0.06 mg/l. over the first 17 days after treatment. The DDT concentrations in drinking water ranged from 0.001 to 0.200 mg/l. To avoid such DDT levels in surface water, the distribution of DDT from aircraft should be discontinued, a safety distance of 1 km from surface waters maintained, and water samples periodically analyzed for DDT residues. DDT will be replaced by other chemicals in 1972.

73-0606. Polizu, A.; Greger, H.; Cojocaru, N. (Plant Protect. Res. Inst., Bucharest, 1, Rumania). **Studies on dinitro-butyl-phenol residues in potato tubers following plant desiccation.** *Z. Pflanzenkr. Pflanzenpathol. Pflanzenschutz 79(6): 343-348; 1972.* (7 references)

In tests conducted between 1968 and 1969 at the Research Institute for Sugarbeet and Potato in Brasov, Dibutox 25% EC, a product based on 2,4-dinitro-6-*sec*-butylphenol (dinoseb, DNBP), was applied to various potato varieties to determine the amounts of DNBP residues penetrating into the potato tubers and their persistence. The earliest sampling of the tubers occurred four days after treatment; for varieties such as Bintje and Colina, this was sufficient time to show desiccation of the stem in most plants even at a rate of 4.5 kg/ha. Five days after treatment, all the plants of these two varieties dried. DNBP residues accumulated in the potato tubers generally ranged between zero and 0.09 ppm. The amounts of toxicant accumulations were directly proportional to the application concentrations and the quantity of active ingredient per hectare. The application of 4.3 kg active ingredient per ha resulted in an accumulation of DNBP residues of up to 0.25 ppm in the rind of the tuber and between zero and 0.05 ppm in the pulp. When stems were removed mechanically prior to treatment, the amounts of residues were usually 2 to 3 times higher.

See also **73-0502** **73-0506** **73-0519** **73-0526** **73-0536** **73-0539** **73-0614** **73-0626** **73-0633** **73-0663** **73-0680** **73-0685** **73-0691** **73-0709** **73-0710** **73-0721** **73-0723** **73-0724** **73-0728**

73-0607. Law, S. E. (Agr. Eng. Dept., Univ. of Georgia, Athens, GA 30601). **Respirators and masks.** *Agr. Eng. 53(11): 12-14; 1972.*

A provision of the Occupational Safety and Health Act which concerns users of pesticides states that no employee dealing with toxic or harmful agents should suffer material impairment of health or functional capacity, even with regular exposure to the hazard for the period of his working life. Air purifying, air supplied, and self-contained respiratory protective devices are used to protect the worker from atmospheres containing toxic particulates and gases. Most mechanical filter respirators protect against particles of approximately 0.3μ, but not against volatile particulate matter typical of many pesticides. A gas or vapor adsorber is used with a high efficiency particulate filter in such situations. Although charcoal and soda lime are used to remove organochlorines, they do not remove organophosphates. Proper fit, maintenance, and cleaning of the masks is essential for protection.

73-0608. McParland, P. J.; McCracken, R. M.; Thompson, R. H. (Vet. Res. Lab., Stormont, North Ireland). **Poisoning on the farm—warfarin.** *Agr. N. Ireland 47(4): 136-137; 1972.*

Warfarin poisoning in domestic animals generally results from careless use, storage, or disposal of the chemical or its container and has increased in recent years. Pigs, calves, dogs, cats, and rabbits are particularly susceptible and poultry is resistant to such poisoning. Ordinary movements such as walking can cause fatal hemorrhage in poisoned animals. Domestic animals generally will consume the bait if available because of its form of a cereal meal. Two and a half ounces of rat bait per day for seven days will kill pigs of approximately 70 pounds. Consumption of poisoned rat carcasses can also result in poisoning and death. If poisoning is suspected, the animals must be kept warm and given an adequate water supply. Handling and surgical procedures should be avoided and obstacles should be removed from pens.

73-0609. Drenth, H. J.; Ensberg, I. F. G.; Roberts, D. V.; Wilson, A. (Dept. of Pharmacol., Univ. of Liverpool, Liverpool, L69 3BX, England). **Neuromuscular function in agricultural workers using pesticides.** *Arch. Environ. Health 25(6): 395-398; 1972.* (3 references)

One hundred and two male agricultural workers who used pesticides underwent electromyographic (EMG) examination for signs of disturbed peripheral nerve and muscle function. In approximately 40%, abnormal EMG patterns were seen, similar to those in methylsulfate and pyridostigmine bromide poisoning and in male pesticide-factory workers exposed to mixed organophosphorus and organochlorine compounds. Fifty-three men were reexamined two months later and in 25 the EMG pattern changed either from abnormal to normal, or vice versa. Concurrent measurement of blood cholinesterase activity showed no difference between men with normal and abnormal EMG records; and all values were within normal limits, as were a number of biochemical blood measurements related to hepatic and renal function. No special causal agents were identified and no dosage-response relationship was established. (Author abstract by permission)

73-0610. Int. Ass. on Occup. Health (Zielhuis, R. L., Coronel Lab., Fac. of Med., Univ. of Amsterdam, Amsterdam, The Netherlands). **Epidemiological toxicology of pesticide exposure.** *Arch. Environ. Health 25(6): 399-405; 1972.*

On behalf of the subcommittee on Pesticides, Permanent Commission, and International Association on Occupational Health, a workshop convened in Amsterdam to discuss epidemiological toxicology of pesticide exposure. Emphasis was put on long-term exposure to organochlorine and/or organophosphorus compounds. The workshop emphasized the need for improving methods of research; phenomena for field study were suggested. It proposed some tentative guidelines for absorption of and response to DDT, aldrin-dieldrin and cholinesterase inhibitors to be used in occupational health programs. Occupational epidemiology appeared to provide essential information to estimate the risks of exposure of the general population. The workshop concluded that observations in workers so far have not yielded evidence that deviations in biological phenomena are likely to occur in the nonoccupationally exposed general populations. (Author abstract by permission)

73-0611. Blaszyk, P. (Pflanzenschutzamt der Landwirtschaftskammer Weser-Ems, 29 Oldenburg, Germany). **Beeintraechtigung der freilebenden Tierwelt durch Pestizide. [Pesticide-induced injuries to wildlife.]** *Ber. Landwirt. 50(2): 404-416; 1972.* (97 references) (German)

A survey of pesticide-induced injuries to wildlife reveals that losses among birds have been extensive while injuries to mammals have occurred less frequently. Animals may die as a direct result of pesticide application or be killed at various intervals after pesticide application by eating contaminated prey. Animals at the tops of food chains which consume a limited variety of highly contaminated foods are particularly endangered. Many reports have attributed the reproductive failures in carnivorous birds to the presence of sublethal quantities of pesticides in their tissues. Renormalization of reproduction rates has been reported from areas where the use of persistent pesticides was discontinued. Improved pesticide residue monitoring systems for wildlife and intensification of testing for effects on wildlife prior to pesticide approval will aid in minimizing wildlife losses.

73-0612. Anonymous. **Toxicity of thallium.** *Brit. Med. J. 3(5829): 717; 1972.* (7 references)

Thallium is an extremely toxic and cumulative poison which can enter the body by ingestion or absorption. Its use in rodenticides and insecticides constitutes its main hazard to public health. Symptoms of poisoning include loss of hair, limb pains, peripheral neuritis, proteinuria, and occasionally neurological signs. Treatment is difficult because fecal and urinary elimination of the metal are slow. Chelating agents are somewhat useful as is potassium chloride. The early use of a screening test for thallium on urine is advised.

73-0613. Boros, S. J. (Group Health West Med. Cent., Minneapolis, MN 55419). **An unusual instance of poisoning due to a glycol derivative. Toxicity from U.S. Army Insect-Repellent Clothing Treatment Solution M-1960.** *Clin. Pediat. 11(6): 364-366; 1972.* (11 references)

A case of poisoning from U.S. Army Insect Repellent Clothing Treatment Solution M-1960 in a 4-yr-old Vietnamese girl is reported. The child became confused and somnolent soon after she ingested about 50 ml of the material. Emesis occurred about 45 min after ingestion. CNS depression was quite pronounced 1 hr later. The patient's chest remained clear and urinalysis was normal. Dramatically positive Babinski responses were observed. Five hr after ingestion, these were negative, the child showed signs of improvement, and recovery followed. Hemoglobin level was 8.5 g and climbed to 13 g after 2 mo of iron treatment. A comparison of the girl's symptoms with those observed during field trials of the formula and with those known to be caused by the various ingredients indicated that the intoxication was largely due to the toxicity of 2-butyl-2-ethyl-1,3 propanediol (ethylbutyl propanediol). Although there was no obvious renal damage, the clinical picture was similar to those of other reported glycol poisonings.

73-0614. Everaarts, J. M.; Koeman, J. H.; Brader, L. (Inst. de Pharmacol et de Toxicol. Vet., Utrecht, The Netherlands). **Contribution a l'etude des effets sur quelques elements de la faune sauvage des insecticides organo-chlores utilises au Tchad en culture cotonniere. [Contribution to the study of the effects of organochlorine insecticides on some elements of the wildlife fauna in cotton plantations in the Republic of Chad.]** *Coton Fibres Trop. 26(4): 385-394; 1971.* (25 references) (French)

Effects of organochlorine insecticides used on cotton fields in Chad were studied on some species of the wildlife fauna. Mortality occurred among herbivorous (mainly seed-eating) and insectivorous birds after spraying cotton fields with endrin-DDT mixtures. Deaths occurred mainly due to endrin, and, in a few cases, to dieldrin. Insectivorous birds already were becoming scarce in cotton areas. The distribution of insecticides was measured by using *Ceryle rudis, Megaceryle maxima, Clarias anguillaris* and *Hydrocynus forskalii* as indicators. The endrin, dieldrin, DDT, DDD (TDE), and DDE residues found in organs of these species were small compared to corresponding concentrations determined in European species. The endrin, dieldrin, DDT, DDD, and DDE concentrations in the livers and brains of *Ptilopachus petrosus, Euplectes orix, Lemniscomys striatus, Uraeginthus bengalus, Turdus pelios, Centropus senegalensis, Crecopsis egregia, Ploceus vitellinus, Spermestes cucullatus, Estrilda* and *Ortygospiza articollis* were in the respective ranges of 0.13-1.42, 0.07-32.5, 0.07-6.85, 0.1-6.98, and 0.15-30.4 ppm. The DDE and endrin concentrations in *Hydrocynus forskalii* and *Clarias anguillaris* were 0.007-0.16 and 0.005-0.083 ppm, respectively. The results indicate no major accumulation of insecticides in Lake Chad and its tributaries. The residue levels in food products (sorghum and fish) were generally well below the maximum allowable concentrations, except for endrin.

73-0615. Luechtrath, H. (Pathologisches Institut, Koblenz, Germany). **Die Leberzirrhose bei chronischer Arsenvergiftung der Winzer. [Liver cirrhosis in vinedressers due to chronic poisoning with arsenic.]** *Deut. Med. Wochenschr. 97(1): 21-22; 1972.* (13 references) (German)

Massive liver cirrhoses in vinedressers in Germany in 1942 were due to chronic poisonings with arsenical insecticides. As the use of such insecticides was banned in 1942, the incidence of liver cirrhosis among vinedressers was sharply reduced and recovery of 49% of all cirrhotic patients with the elimination of the poison from the body was observed. A recent wave of liver cirrhosis, not only among vinedressers, is due to chronic hepatitis and alcohol abuse rather than to arsenic.

73-0616. Klimmer, O. R. (Toxikol. Abt., Pharmakol. Inst. der Univ., 53 Bonn, Germany). **Insekten- und Fliegenschutzpraeparate. [Insect and fly control preparations.]** *Deut. Med. Wochenschr. 97(15): 634; 1972.* (German)

The problem of the possible hazards involved in the indoor use of insecticides and especially of fly control preparations such as fly strips is reviewed. The use of fly strips is not hazardous to health due to the very low, ineffective concentration of the insecticide emitted. However, any contact of such preparations with foodstuffs should be avoided, especially because fly strips may contain persistent, accumulating organochlorine insecticides as the effective agent.

73-0617. Hofmann, A.; Frohberg, H. (Inst. fuer Toxikologie, E. Merck, Darmstadt, Germany). **Gramoxone-Intoxikationen in der Bundesrepublik Deutschland. [Paraquat poisonings in Germany.]** *Deut. Med. Wochen-*

schr. 97(35): 1299-1303; 1972. (51 references) (German)

Thirty-eight cases of accidental and intentional poisonings with Gramoxone (paraquat) in Germany are analyzed. The poisonings were lethal in 7 of the 18 accidental, and in 16 of the 20 intentional poisonings. The intake of large amounts (about 100 mg) of paraquat resulted in lesions of the oropharynx, vomiting, diarrhea, massive kidney insufficiency with azotemia, alveolar and interstitial lung edema, liver damage, and finally in death in 1-3 days after intake. A latency of 1-2 days observed in cases of intakes of 10-40 ml was followed by tachypnea, kidney insufficiency with oliguria, increased serum urea and creatinine levels, slight liver damage with icterus, and increased transaminase activity. In cases when the kidney function failed to recover, death occurred within 10 days due to acute inflammatory lung damage. With kidney insufficiency at least functionally reversible, lung fibrosis and dyspnea may develop in two weeks after intake and lead to death within 3 weeks. Benign, transitory alterations in the lung function in cases with kidney function recovered are observed. While there is no specific antidote for paraquat available, the absorption of the poison by Fuller's earth or bentonite, followed by forced osmotic diuresis for several days in intensive care facilities, are imperative. Dialysis is not effective due to the low paraquat concentration in the blood, and oxygen therapy should be avoided due to the assumed formation of peroxide in the lung.

73-0618. Ishikawa, T. (Sch. of Ophthalmol., Fac. of Med., Kitasato Univ., Mokadai, Sagamihar, Kanagawa Prefect., Japan). **[A plan for setting up a standard of diagnosis of chronic organophosphorus poisoning due to agricultural chemicals.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9): 817-820; 1972.* (Japanese)

The first screening for organophosphorus poisoning in children checks for deterioration of eyesight, loss of equilibrium, extreme weakness in physical training, very nervous or backward children, remarkably thin children or those gaining little weight. The number of children proving positive to those five items is now almost 1 or 2 out of 100 in rural districts in Japan. The second screening, administered by an ophthalmologist, provides the major information for diagnosis. Deterioration of eyesight, narrowness of vision, myopia, strong astigmatism of both eyes, loss of equilibrium, decrease of cholinesterase, efficacy of PAM or Padrin (pyrodifenium bromide), and acceleration of knee reflex are studied. A case is confirmed positive if more than five items out of eight are present. Subsidiary symptoms for diagnosis include atrophy of the optic nerve, subnormal ERG, poor condition of the pupil, depression of the leg reflex, depression of the autonomic nervous system, and altered liver function.

73-0619. Takaku, I. (Dept. of Ophthalmol., Nagasaki Univ., Sch. of Med., Nagasaki, Nagasaki Prefect., Japan). **[Studied report of children who have eye diseases due to organophosphate pesticides in Nagasaki Prefecture.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9):820-822; 1972.* (Japanese)

The findings are given on a study by the Nagasaki University Department of Ophthalmology on 22 subjects with organophosphate related eye disorders. The subjects ranged in age from six to twelve years. Screening criteria included myopia and astigmatism not corrected by glasses and narrowness of vision. Results of eye examinations revealed that thirty-two eyes were subnormal. Six eyes had damaged retinas. Twelve eyes had extremely narrow vision, 12 had fairly narrow vision, and 11 eyes had slightly narrow vision. In another study the results of treatment with Padrin (pyrodifenium bromide) are reported for 152 women and 63 men with agricultural chemical related eye disorders. The subjects were divided into three groups for experimental treatment. The first group were given only Padrin for one month. The second group were given Padrin for one month and then given mydrin. The third group were given Padrin and mydrin like the second group and then treated with Padrin again. According to the findings, the recovery of eyesight was mainly due to the action of Padrin.

73-0620. Mitsui, Y.; Tamura, O. (Dept. of Ophthalmol., Tokushima Univ., Sch. of Med., Tokushima, Tokushima Prefect., Japan). **[Report of children with eye diseases due to organophosphate pesticides in Tokushima Prefecture.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9): 822-824; 1972.* (Japanese)

A hundred and thirty-one children of kindergarten and primary school age were chosen for group examination. They were from an area in Tokushima where organophosphate chemicals were the most widely used and where organophosphate pesticides were detected from dust of the houses and sheds. The method of examination included examination of eyesight, refraction, vision, eyeground, and equilibrium, tested by standing on one leg. Abnormal symptoms in eyesight and refraction occurred in 19 subjects; narrowness of vision occurred in 26 subjects; abnormal conditions of eyegrounds occurred in 8 subjects; loss of equilibrium occurred in 2 subjects. Fourteen subjects out of the total 64 showed two of the above conditions.

73-0621. Otsuka, J.; Tokoro, T. (Tokyo Ikashika Univ., Dept. of Ophthalmol., Bunkyo, Tokyo, Japan). **[Study on a standard of diagnosis of eye diseases due to organophosphate pesticides from the viewpoint of eye refraction.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9): 825-828; 1972.*(Japanese)

Children poisoned with organophosphate pesticides showed symptoms of astigmatism and cholinesterase-related optic-axis length. The less cholinesterase in blood serum, the longer the optic-axis. Cramp of ciliary muscle in the first stage of poisoning and subsequent relaxation and extension of optic axis were supposed to occur.

These theories were tested in animals. Dogs poisoned with organophosphate pesticides were myopic in comparison with normal dogs; the myopia was supposedly caused by the previously mentioned phenomena. Treatment with mydrin was given. Intraocular pressure of poisoned dogs was lower before mydrin was applied and higher than that of normal dogs after application.

73-0622. Saito, M.; Kubo, H. (Dept. of Clinical Pathol., Kitasato Univ., Sagamihara, Kanagawa Prefect., Japan). **[Report of children with eye diseases due to organophosphate pesticides from a biochemical point of view.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol). 66(9): 828-831; 1972.* (Japanese)

Diagnosis of eye diseases was done by measuring cholinesterase activity. In 67 normal subjects, the true cholinesterase value was 2.04 ± 0.19 $\mu M/\mu l$/min and pseudo cholinesterase was 5.57 ± 0.95 $\mu M/\mu l$/min. In 41 patients from Kitasato University, Department of Ophthalmology, true Che was 1.67 ± 0.12 $\mu M/\mu l$/min and pseudo ChE was 6.09 ± 0.92 $\mu M/10\mu l$/min. The true ChE values of 450 subjects in an area contaminated by agricultural chemicals in 74 normal adults were 1.56 ± 0.34 $\mu M/\mu l$/min. In 51 organophosphate poisoned women the values were 1.46 ± 0.29 $\mu M/\mu l$/min. Whereas 39 normal children had values of 1.83 ± 0.30 $\mu M/\mu l$/min, 61 secondary school children who complained of deterioration of eyesight had 1.53 ± 0.25 $\mu M/\mu l$/min. Fifty-two untreated organophosphorus poisoning patients had values of 1.26 ± 0.28 $\mu M/\mu l$/min, while 74 patients treated with Padrin (pyrodifenium bromide) had 1.42 ± 0.27 $\mu M/\mu l$/min, and 47 patients treated with Padrin and PAM had 1.45 ± 0.36 $\mu M/\mu l$/min. Analysis of organophosphate pesticides in agricultural chemicals by gas chromatography is another method of diagnosis. The graph drawn as a result of analysis of a normal person's urine by means of GC was quite different from the one of urine of organophosphorus poisoning patients.

73-0623. Ohba, N.; Mishima, S.; Ohdo, K. (Tokyo Univ. Sch. of Med., Dept of Ophthalmol., Tokyo, Japan). **[Visual field anomalies in Japanese juveniles from the vicinity of Saku, Nagano Prefecture, Japan.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9): 835-837; 1972.* (3 references) (Japanese)

Elevated incidences of visual field anomalies have been reported in children from several areas in Japan. Visual field examinations were performed on 19 children, 7-14 years old, presumed to be victims of chronic organophosphate intoxication; 10 normal adults, 20-40 years old; and 5 normal children, 7-11 years old. The threshold intensity for sighting was measured using the Goldman perimeter at various retinal angles with respect to the right eye, ranging from 0°(foveal vision) to 60° in 10° increments, to the nasal and temporal sides in the horizontal plane of the visual field. The variation in threshold intensity among the presumably pesticide-exposed children was much greater than in the normal adults (2 log units Apostilb compared to 0.5 log units); however, a rather broad variation range was also found in the presumably normal children. Measurements in two normal children 11 and 7 years of age were compared with measurements in two children 8 and 14 years of age with clinically diagnosed organophosphate poisoning. The threshold intensities in the poisoning cases were distinctly higher than those of the corresponding-age normal. However, standards for visual field parameters in normal children must be reliably established before meaningful comparisons can be made.

73-0624. Tsukahara, I.; Fukuchi, S.; Hattori, M. (Kansai Med. Univ., Sch. of Ophthalmol., Japan). **[Examination for chronic pesticide poisoning based on examinations of visual acuity, eyegrounds, and ERG.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9): 838-848; 1972.* (5 references) (Japanese)

In the course of studies on eye damage due to chronic pesticide poisoning, ophthalmologic examinations were performed on three groups of children. Group A included 23 children, normal except for mild conjunctivitis; Group B, 18 children with chronic exposure to pesticides; and Group C, 17 children with depressed erythrocyte/serum cholinesterase ratios. Visual acuity could be corrected with lenses in all children in Group A and all but one in group C; however, visual acuity in one or both eyes could not be corrected to normal values in 7 of the children in Group B. Eyegrounds of the Group A children were normal. In 11 children from group B, the papillae at the nasal margin were indistinct and slight congestion and opacity of the papillae were apparent at the sides of the eyes nearest the nose. Slight to severe discoloration of the papillae was visible in 5 others. The findings were identical bilaterally. In group C, 9 children displayed symptoms resembling those of papillitis. No abnormalities were observed in the electroretinograms from the various groups which could be correlated with the other eye findings in these children.

73-0625. Abdel Kader, M. M.; Emara, A. M.; Kamel, G.; Habib, A.; El-Samra, G. H.; Abdel-Halim, S. (Dept. of Biochem., Cairo Univ., Cairo, Egypt). **Chemico-pathological changes in the liver of industrial workers chronically exposed to cotton dust.** *J. Egypt. Med. Ass. 54: 489-501; 1971.* (38 references)

Men chronically exposed for at least four years to cotton dust (DDT, lindane, and sulfur in a ratio of 10:3:40) were divided into two groups—those who did and those who did not have enlarged livers. A control group was obtained from a non-hazardous plant. In the group having enlarged livers, a statistically significant drop in serum total proteins and serum albumin was observed, but no marked change occurred in serum $alpha_2$ and beta-globulins. Those men without enlarged livers had significantly lower serum $alpha_2$ and beta-globulins, but not lower serum total proteins or serum

albumin. Both groups had levels of serum alpha$_1$ globulin, serum gamma globulin, and SGPT similar to those of controls. An insignificant elevation of SGOT and decrease of pseudocholinesterase values were noted in both groups. Liver biopsies obtained from five men with the most pronounced symptoms showed fatty degeneration of liver cells, cloudy swelling, necrotic changes, and sometimes cellular infiltration with polymorphs, monocytes, lymphocytes, plasma cells, and fibroblasts. No correlation was obtained between length of exposure and degree of liver enlargement.

73-0626. Flickinger, E. L.; King, K. A. (Bureau of Sport Fisheries and Wildlife, Victoria, TX). **Some effects of aldrin-treated rice on Gulf Coast wildlife.** *J. Wildl. Manage. 36(3): 706-727; 1972.* (29 references)

A total of 192 bird casualties, presumed to represent a much larger number, were collected between 1967 and 1971 from three Texas coastal areas planted with aldrin-treated rice seed. Aldrin or dieldrin residues were found in all these birds (up to 16 ppm in whole bodies) and in all eggs, scavengers, predators, fish, frogs, invertebrates, and soil tested. Endrin and DDT were the next most frequent residues and mercury was found in all 24 samples analyzed. Mercurials, which are still employed, had been used about five years prior to the introduction of aldrin without a noticeable effect on bird populations. Some birds were exposed to the organochlorines by ingestion of treated seed, but many of the poisoned species fed largely on invertebrates. When fulvous ducks, highly susceptible to aldrin-dieldrin poisoning, were exposed experimentally for three days to fields aerially planted with treated seed, 3 of 10 birds died and others were intoxicated. Brain residues in birds that died were 2.5, 2.9, and 6.8 ppm dieldrin. Ducks penned in fields planted with untreated seed did not die and some gained weight.

73-0627. Koch, R.; Winter, R.; Tillmann, P.; Wiessmann, B. (Medizinische Klinik und Poliklinik, Westfaelische Wilhelms-Universitaet, Muenster, Germany). **Forcierte Diurese bei Thalliumvergiftung. [Forced diuresis in the treatment of thallium poisoning.]** *Med. Welt 23(17): 649-651; 1972.* (18 references) (German)

Treatment in a case of intentional poisoning with 507 mg of thallium is described. It was possible to increase the rate of thallium elimination with the urine to 3.5 times the normal value by forced diuresis. With urine volume increased to 12 l./day, 62% of the total thallium absorbed was eliminated in 10.6 days. Urine samples taken in the fourth week of the treatment contained no more thallium. The urine thallium contents were determined by atomic absorption spectroscopy.

73-0628. Loew, H.; Tillmann, P.; Winter, R.; Wiessmann, B.; Koch, R.; Schiller, M. (Medizinische Universitaets-Poliklinik, Muenster, Germany). **Thallium-Elimination durch die Haemodialyse im Vergleich zur grossen Diurese bei einer schweren Thallium-Intoxikation. [Thallium removal by hemodialysis in comparison with copious diuresis in severe thallium intoxication.]** *Med. Welt 23(41): 1411-1412; 1972.* (3 references) (German)

Treatment in a severe intentional poisoning with 1.3-1.8 g of thallium sulfate is described. Obstipation, paresthesia, hyperesthesia, pains in the legs, motor paralysis, tachycardia, and loss of hair were the principal symptoms of the thallium poisoning. Forced diuresis yielding about 12 l. of urine per day over several days, immediate continuous hemodialysis at a rate of 500 mg/min in a single-pass system, as well as laxatives, and Prussian blue with daily clysters were applied. Hemodialysis was superior to forced diuresis while simultaneous hemodialysis failed to reduce the effect of the forced diuresis.

73-0629. Krupa, A.; Pienkowska, H.; Tarka, Z. (Lublin Regional Toxicology Center, Lublin, Poland). **Ciezkie zatricie tiuramem. [Acute poisoning with "thiram."]** *Med. Wiejsk. 6(1): 29-31; 1971.* (7 references) (Polish)

A 48 year old agricultural worker was hospitalized with vomiting, diarrhea, dizziness, weakness, and severe headache six hours after having mixed thiram with water for 10 minutes. He also had ingested some whiskey the previous evening. Laboratory tests revealed: hemoglobin 106%, RBC 5,500,000, hematocrit 53 V%, WBC 11,200. Urine contained albumin and urobilinogen and numerous casts. On the second day after admission CO_2 = 37 V% and transaminase activity increased: SGOT 102 units, SGPT 40 units. On the fourth day after exposure a severe ulceration of oral cavity, decrease of cholinesterase activity to 1/3 of normal, and liver enlargement were noted. The ulceration of mucous membrane and liver enlargement persisted for three weeks in spite of intensive symptomatic treatment (infusion of 5% glucose, physiologic NaCl, multiple electrolyte fluid, large doses of vitamin C, antazoline, cardiamide, caffeine, oxyterrazine, hepazon, mycostatin, and painting of oral cavity with antiseptics. Alkali levels were normalized with a single administration of tromethamine.

73-0630. Hays, H.; Risebrough, R. W. (Author address not given.) **The early warning of the terns.** *Natur. Hist. 80(9): 39-47; 1972.*

A study of the breeding biology of two species of terns on Great Gull Island in Long Island Sound uncovered an increasing number of abnormalities which may be indicative of the threat to human health from various pollutants in the Sound. Bill and leg deformities, missing flight feathers, and thin-shelled eggs were observed in the colony. Levels of DDT in young birds and in fish brought to the colony by adults were no higher than in many other bird populations although samples from

1971, when shell-thinning was noted, have not been analyzed. Relatively high levels of polychlorinated biphenyls (PCBs) were found in birds and fish. Recent research has demonstrated the teratogenic nature of contaminants in PCB. High priority should be given to a study of the pollutants present in Long Island Sound. Technological direction should be changed to meet the crisis caused by exploitation of the natural systems.

73-0631. Szabo, S. (Inst. de Med. et de Chirurgie Experimentales, Univ. de Montreal, Montreal, Canada). **A katatoxikus steroidokrol. [On catatoxic steriods.]** *Orv. Hetil. 113(26): 1515-1518; 1972.* (107 references) (Hungarian)

Studies on catatoxic steroids are reviewed. Catatoxic steriods exert their nonspecific antitoxic effect through inducing or activating hepatic microsomal enzymes or through extramicrosomal and/or extrahepatic detoxication by promoting excretion or depot formation. Catatoxic steriods are effective antidotes for such pesticides as ethion, dioxathion, EPN, azinphosmethyl, and parathion. A DDT derivative, TDE, may be effective in the treatment of Cushing's syndrome.

73-0632. Leng, J.-J.; Greenblatt, R. B. (Faculte de Med. de Bordeaux, Bordeaux, France). **Hirsutism in adolescent girls.** *Pediat. Clin. N. Amer. 19(3): 681-703; 1972.* (44 references)

The fungicide hexachlorobenzene is one of a number of agents which have hirsutism (excessive hair growth) as a side effect. Forty-two patients from 10 to 19 yrs of age among 348 exposed to this drug acquired toxic porphyria cutanea tarda. Frequently, non-sexual hair growth was excessive on chin, face, and extremities and sometimes the entire body was covered with hair, causing the condition to be labeled "monkey disease".

73-0633. Coppage, D. L.; Duke, T. W. (Gulf Breeze Lab., Environmental Protection Agency, Gulf Breeze, FL 32561). **Effects of pesticides in estuaries along the Gulf and Southeast Atlantic coasts.** *Proceedings of the 2nd Gulf Coast Conference on Mosquito Suppression and Wildlife Management, 1971, 24-31.* (36 references)

Some of the findings on residues of persistent pesticides in estuaries and some effects of these pesticides observed in field and laboratory studies are reviewed; results of a recent investigation on the effects of malathion are presented. DDT, dieldrin, endrin, aldrin, BHC-lindane, heptachlor, toxaphene, and mirex have been found in surface water, sediment, or aquatic life in estuaries along the Gulf and Southeast Atlantic coasts. In some Louisiana estuaries, organochlorine residues, particularly dieldrin, appear to be declining. Biological magnification of DDT residues has been documented and there is evidence that these residues interfere with fish reproduction. Estuarine species exposed experimentally to levels of some persistent pesticides currently present in estuarine waters show effects such as reduced growth, reduced reproduction, and mortality. Organochlorines will probably be replaced with the less persistent carbamates and organophosphates which inhibit brain AChE. A study presently underway has determined the relationship of brain AChE of spot (*Leiostomus zanthurus*) to malathion concentration in water, length of exposure, and death. The mean reduction of activity for all lethal exposures was 81%. Fish from two Louisiana lakes in areas sprayed with the pesticide for control of mosquitoes exhibited reductions of brain AChE as low as 82%.

73-0634. Luzhnikov, Ye. A.; Shimanko, I. I.; Aleksandrovskiy, V. N. (Moscow N. V. Sklifosovskiy Sci. Res. Inst. of First Aid, Moscow, USSR). **Differentsial'nyy diagnoz i neotlozhnaya terapiya komatoznykh sostoyaniy pri nekotorykh ostrykh otravleniyakh. [Differential diagnosis and emergency therapy of some comatose states consequent to acute poisonings.]** *Sov. Med. 35: 16-22; 1972.* (4 references) (Russian)

Principles of differential diagnosis and emergency therapy of comatose states in acute poisonings are presented. The major groups of acute poisonings accompanied by comatose conditions (poisonings with organophosphorus pesticides, carbon monoxide, alcohol, and barbiturates) have common clinical pictures. Electroencephalographic and rapid toxicological tests are of great value in identifying the poison for correct therapy. The emergency therapy of acute poisonings includes the elimination of the poison from the body, antidotal treatment, and symptomatic therapy. Suitable antidotes against organophosphorus pesticides are pralidoxime and TMB-4. Unithiol, prozerine, and vitamin B1 are useful as antidotes for other poisons.

73-0635. Naydenov, N.; Stoyanov, Kh.; Yotov, N. (IBPRNB, Sofia, Bulgaria). **Khronichno otravyane na kravi s organofosforni insektitsidi. [Chronic poisoning with organophosphate insecticides in cows.]** *Vet. Sb. 69(9): 44-45; 1972.* (Bulgarian)

A cow sickness, attributed to chronic poisoning with organophosphates, is described in two groups of cows from areas located close to Pazardzhir in Bulgaria. Symptoms included loss of appetite, diarrhea, salivation, and increased pulse rate. Increased peristalsis and decreased amount of milk production (from 16-18 to 2-3 l./day) constituted other symptoms. The body temperature was normal, but whole blood cholinesterase activities were 54% lower and blood sugar was 12% higher than in clinically healthy cows. Successful specific treatment including atropine (1% solutions, administered s.c. and i.v.) followed by 10 mg/kg of PAM shortly thereafter confirmed the diagnosis. Fodder, hay, and grass analysis indicated the presence of 1 to 3 mg/kg of cholinesterase inhibitor residues, and further infor-

mation helped conclude that the forage given to the cows during the past six months was contaminated with phosthiol (fostiol).

73-0636. Meyer, F. P.; Staib, A. H. (Abteilung fuer klin. Pharmakol., Inst. fuer Pharmakol. und Toxikol. der Med. Akad. Magdeburg, Magdeburg, DDR). **Ueber die Vergiftungen mit Phosphorsaeureestern. [On poisonings with phosphoric acid esters.]** *Z. Aerztl. Fortbild. 65(1): 1-3; 1971.* (24 references) (German)

Ten cases of accidental poisoning with methyl parathion, dimethoate, trichlorfon, methyl demeton, and dichlorvos are described. Mostly irreversible inhibition of the acetylcholinesterase activity, liposolubility, penetration also into the central nervous system, and low rate of elimination are common features of most of the phosphoric acid esters. Diarrhea, salivation, nausea, headache, cough with increased rate of bronchial and tracheal secretion, bronchial constriction, bradycardia, miosis, cyanosis, pulmonary edema, coma, tonic-clonic spasms, tremor, and death due to asphyxiation are the regular symptoms of poisoning in cases of varying seriousness. Forced vomiting, gastric lavage, and antidote therapy are imperative. Liberal i.v. administration of atropine may be necessary for several days. The pralidoxime or obidoxime dose, to be administered during atropine therapy, should not exceed 10 mg/kg. Pralidoxime and obidoxime should not be used in cases of dimethoate poisoning.

73-0637. Goldhammer, Y.; Sarova-Pinhas, I.; Braham, J. (Dept. of Neurol., Chaim Sheba Med. Cent., Tel-Hashomer, Tel Aviv Univ. Med. School, Israel). **Persistent coma and papilledema in parathion poisoning.** *Z. Neurol. 202(2): 151-153; 1972.* (6 references)

A case of parathion poisoning in which papilledema associated with edema of the brain and hemiplegia were observed is described. These symptoms have been reported infrequently with parathion and not at all with other organophosphates such as TEPP. Treatment with diuretics, digitalis, oxygen, atropine, and steroids failed to induce a change in the comatose state of the patient. A right frontotemporal decompression was done but there was little change in condition and the youth died after two months.

See also 73-0528 73-0604 73-0707

73-0638. Liebmann, H.; Matthies, H.; Kumbier, E. (Inst. Pharm. Toxikol., Med. Acad., Magdeburg, DDR). **Der Einfluss von Phenelzin auf die durch Reserpin veraenderte Toxizitaet cholinerger Pharmaka. [The influence of phenelzine on the reserpine-modified toxicity of cholinergic drugs.]** *Acta Biol. Med. Ger. 26(3): 551-558; 1971.* (9 references) (German)

The reserpine-induced alterations in toxicity of cholinergic drugs, demonstrated in previous studies, suggested an influence of the adrenergic nervous system on the cholinergic nervous system via the biogenic amines. Phenelzine, a monoamine oxidase inhibitor, antagonizes the effects of reserpine in vivo; therefore, the effect of phenelzine on the reserpine-induced enhancement of cholinergic drug toxicity was studied in mice. Although the toxicity of DFP and cholinergic drugs was appreciably enhanced by administration of reserpine alone, the toxicity of paraoxon was insignificantly changed. Phenelzine alone enhanced the toxicity of all substances tested, except paraoxon, only within the first hour after administration. Administration of phenelzine 4 hr before reserpine administration prevented the enhancement of cholinergic-drug toxicity as demonstrated for DFP and other cholinergics. Paraoxon toxicity remained essentially unchanged. The findings with ganthamine were similar to those with paraoxon; the reasons for the differences between these two substances and the others tested were not apparent. The toxicity seems to be enhanced primarily by norepinephrine depletion; the role of serotonin has not been elucidated.

73-0639. Kleinrok, Z.; Malec, D. (Dept. of Pharmacodynamics, Med. Acad., Lublin, Poland). **Effects of sodium diethyldithiocarbaminate on uptake of oxygen by rat brain, liver, and kidney slices.** *Acta Pol. Pharm. 28(4): 444-448; 1971.* (20 references)

DEDTC in concentrations of 0.05, 0.1, and 0.2% reduced oxygen uptake in vitro in rat liver, brain, and kidney slices. Significant reductions were observed only with the highest concentrations of DEDTC applied to brain slices and with each concentration applied to liver slices. In contrast in vivo administration of the compound resulted in an enhancement of oxygen uptake. A significant elevation was observed in liver when the animals were dosed with 200 or 400 mg/kg i.p. Significant effects were not observed on brain or kidney. Intraventricular application of DEDTC at 20 mg/kg caused a significant elevation of oxygen consumption in brain tissue.

73-0640. Ponchet, J.; Tramier, R. (Station de Pathol. Vegetale, Inst. Nat. de la Rech. Agron., Antibes, France). **Effets du benomyl sur la croissance de l'oeillet et la microflore des sols traites. [Effects of benomyl fungicide on the growth of carnation and on the soil microflora.]** *Ann. Phytopathol. 3(3): 401-406; 1971.* (4 references) (French)

Effects of benomyl on the growth of carnation and on the soil microflora were studied. Benomyl had no phytotoxic effect on carnation and did not upset the biological equilibrium of the soil microflora. No modifications occurred before one or two months of treatment, whatever the doses used. The total number of fungi increased slightly due to small doses while single doses of 15 g/m^2 and another dose of 2 g/m^2, repeated every fortnight, decreased the fungi count. No change in populations of *Fusarium, Penicillium, Trichoderma, Gliocladium* and *Chaetomium* as well as of *Actinomycetes* and bacteria occurred. Benomyl had selective effect on *Alternaria, Stemphylium, Stysanus, Stachybotrys, Mucor* and on some species of *Aspergillus (A. ustus)* and *Penicillium.* Proliferation of *Alternaria* and *Stysarus* was observed in some soils.

73-0641. Mostafa, I. Y.; Fakhr, I. M. I.; Bahig, M. R. E.; El-Zawahry, Y. A. (Dept. of Radiobiol., Atomic Energy Authority and National Res. Cent., Cairo, Egypt). **Metabolism of organophosphorus insecticides. XIII. Degradation of malathion by *Rhizobium* spp.** *Arch. Mikrobiol. 86(3): 221-224; 1972.* (15 references)

Rhizobium leguminosarum and *Rhizobium trifolii,* two soil microorganisms, metabolized ^{32}P-malathion at respective rates of 67% and 87% in one week. The identified hydrolytic products were inorganic phosphates, malathion-diacid, malathion-monoacid, dimethyl phosphate, *O,O*-dimethylphosphorothioate, and *O,O*-dimethylphosphorodithioate. The products indicate a very active carboxyesterase system, but the malathion diacid is probably the result of another enzyme which hydrolyzes the second carbethoxy group of the monoacid. Malaoxon was found in negligible amounts in extracts from cultures of *R. leguminosarum* and not at all in those of *R. trifolii,* suggesting that these microorganisms lack the oxidative systems necessary for oxidation of the malathion molecule.

73-0642. Lohs, K. (Forschungsst. fuer Chem. Toxikol., Deut. Akad. der Wissensch., Leipzig, DDR). **Zu aktuellen Problemen der Toxikologie einiger Pflanzenschutz- und Schaedlingsbekaempfungsmittel. [Current problems in pesticide toxicology.]** *Arch. Pflanzenschutz 7(6): 435-449; 1971.* (German)

With increasing public and scientific awareness of environmental problems, toxicologic information needs have become more complex. A simple statement of the acute LD50 value is no longer adequate; it is now recognized that long-term, low-level exposure to toxicants is more likely to occur than acute exposure. The complexities of toxicologic investigation are discussed on the basis of the organophosphate pesticides with particular reference to the metabolic processes which these compounds undergo in vivo and in vitro.

Despite the accumulation of information on structure-activity correlations for these compounds, it is still impossible to predict the reactivity of a new organophosphate compound on the basis of its structure. Organophosphorus compounds can undergo hydrolysis at various sites, several oxidation reactions, formation of "onium" salts, and isomerization reactions; all these processes might proceed simultaneously at different rates. The development of the organophosphate insecticides also stimulated the search for antidotes, the most effective at present being the oxime-type cholinesterase reactivators.

73-0643. Domsch, K. H. (Inst. fuer Bodenbiol., Forschungsanstalt fuer Landwirt., 33 Braunschweig, Germany). **Einfluss von Pestiziden auf mikrobielle Prozesse und oekologische Beziehungen im Boden. [Effects of pesticides on microbial processes and ecological relationships in soil.]** *Ber. Landwirt. 50(2): 392-403; 1972.* (68 references) (German)

The action mechanisms of the various pesticide classes provide indications concerning the probability of effects on microorganisms. Thus fumigants, fungicides, and herbicides, which influence fundamental biological processes, are more likely to affect soil microorganisms than substances which influence higher specific functions of plants or insects (plant tissue differentiation, photosynthesis, cholinesterase, or electrophysiological activity of the nervous system). Although pesticides rarely reduce the total microbial population of treated soil, they may alter the species composition. Catabolic and anabolic process in microbial metabolism are less likely to be affected by pesticides than are the energy-supplying processes such as nitrification. The microbial transformation of inorganic compounds and trace elements has been incompletely studied. The frequently observed increase in available trace elements, sometimes reaching the phytotoxic range, after soil fumigation is unexplained. Little is known as yet about the effects of pesticides on interactions between soil microorganisms; i.e., between parasites and host plants, between predatory organisms and their prey, and between symbiotic organisms and their hosts.

73-0644. Kaul, R.; Bieniek, D.; Klein, W. (Inst. fuer oekologische Chemie, Gesellschaft fuer Strahlen-und Umweltforschung m.b.H., Munich, Germany). **Beitraege zur oekologischen Chemie 46. Isolierung und Identifizierung von Metaboliten des ^{14}C-*trans*-Chlordans aus Weisskohl. [Contributions to ecological chemistry No. 46. Isolation and identification of ^{14}C-*trans*-chlordane from white cabbage.]** *Chemosphere 1(4): 139-142; 1972.* (9 references) (German)

Leaves of ten white cabbages were treated with 10 mg of ^{14}C-labeled *trans*-chlordane. The metabolites obtained were isolated and identified by thin-layer and gas chromatography as well as by spectroscopy. Largely equal proportions of three different metabolites were determined after four weeks with predominance of one of these metabolites after 10 weeks. All metabolites were more hydrophilic than *trans*-chlordane. One of the metabolites was present in too small a quantity to be identified. The two other metabolites were identified as chlordane chlorohydrin and $C_{10}H_7Cl_7O_2$.

73-0645. Goto, M.; Hattori, M.; Miyagawa, T. (Dept. of Chemistry, Gakashuin University, Toshima, Tokyo, Japan). **Beitraege zur oekologischen Chemie. Toxizitaet von alpha-BHC in Maeusen. [Contributions to ecological chemistry. Toxicity of alpha-BHC in mice.]** *Chemosphere 1(4): 153-154; 1972.* (2 references) (German)

The effects of alpha, beta, and gamma (lindane) isomers of BHC, of their metabolites, of 1,2,4-trichlorobenzene, 2,3,5-trichlorophenol, and 2,4,5-trichlorophenol were studied on CR-JC strain male mice. After the animals were fed 600 ppm of the above compounds, they were sacrificed after 12 weeks. Alpha-BHC caused strong hepatomegalia; liver tumors of 0.5-1.5 cm were detected on several occasions. The findings indicate that alpha-BHC or its metabolite is most probably carcinogenic.

73-0646. Wallnoefer, P. R.; Safe, S.; Hutzinger, O. (Bayerische Landesanstalt fuer Bodenkultur und Pflanzenbau, Munich, Germany). **Die Hydroxylation des Herbizids Karsil (*N*-(3,4-dichlorophenyl)-2-methylpentanamid) durch *Rhizopus japonicus*. [Hydroxylation of the herbicide Karsil (*N*-(3,4-dichlorophenyl)-2-methylpentanamide) by *Rhizopus japonicus*.]** *Chemosphere 1(4): 155-158; 1972.* (4 references) (German)

The hydroxylation of the herbicide Karsil (*N*-(3,4-dichlorophenyl)-2-methylpentanamide) in *Rhizopus japonicus* culture containing 12.5 mg of Karsil per liter was studied. The metabolite obtained was identified by mass spectrometry and nuclear resonance spectrometry. First traces of the metabolite, identified as *N*-(3,4-dichlorophenyl)-3-hydroxy-2-2-methylpentanamide, appeared shortly after the beginning of the exponential fungal growth phase; the metabolite contents in the culture gradually increased with time. The mycelium retained 10-20% of unaltered Karsil while 40 n*M* of Karsil per liter was converted into 22 n*M* of metabolite per liter.

73-0647. Hansen, J. C. (Inst. of Hyg., Univ. of Aarhus, Denmark). **The effect of some sulphur and mercury containing fungicides on bacteria.** *Chemosphere 1(4): 159-162; 1972.* (3 references)

Eleven fungicides were tested for toxicity to 211 strains of bacteria isolated from various soils, marine and freshwater sediments, and out-door dust. A marked inhibitory effect was observed in most cases and within each group represented. An obvious difference was

noted between groups. Captan appears to pose no risk to a bacterial population since most strains were resistant or relatively resistant to it. Dithiocarbamates had more pronounced antibacterial activity, particularly to Gram-positive organisms. Mercurials had a similar effect and some pseudomonads were especially resistant to them.

73-0648. Moeschlin, S. (Fac. de Medicine, Universite de Bale, Basel, Switzerland). **Quelques aspects du traitement actuel des intoxications. [Some contemporary aspects of the treatment of poisonings.]** *Chem. Rundsch. 25(11): 297; 1972.* (1 reference) (French)

General aspects and problems in the treatment of poisoning by chemicals are reviewed. Cobalt-EDTA (dicobalt ethylenediaminetetraacetic acid) is an effective antidote for hydrogen cyanide. Cholinesterase-blocking chemicals including some pesticides are antagonized by atropine, pralidoxime, and toxogonin (obidoxime). Pralidoxime and toxogonin must not be administered before atropinization, and, unlike atropine, they must not be given at excessive dosages. No specific antidote for thallium sulfate has yet been found. Pesticides such as paraquat, diquat, and morfamquat attack the mucosa, liver, and kidney after rapid resorption. Adsorbents such as Fuller's earth (500-1,000 ml of 30% suspension) and bentonite (administered in 5-7% suspension) are useful.

73-0649. Ahmad, S.; Knowles, C. O. (Dept. of Entomol., Univ. of Missouri, Columbia, MO 65201). **Metabolism of *N'*-(4-chloro-*o*-tolyl)-*N,N*-dimethylformamidine (chlorphenamidine) and 4'-chloro-*o*-formotoluidide by rat hepatic microsomal and soluble enzymes.** *Comp. Gen. Pharmacol. 2(6): 189-197; 1971.* (12 references)

Chlorphenamidine (chlordimeform) was metabolized by the rat hepatic microsomal fraction and peak activity obtained at pH 7.4 in the presence of 2 μmoles NADPH. The major product was the *N*-demethyl derivative, demethylchlorphenamidine. Other products included 4-chloro-*o*-toluidine, 5-chloroanthranilic acid, and at least four unknown compounds in the organic solvent fraction. The degradation was inhibited by SKF-525A, but not by DFP. Only slight enzymatic degradation of chlorphenamidine occurred in the soluble liver fraction, but the 4'-chloro-*o*-formotoluidide, produced non-enzymatically, was metabolized by this fraction. This product was also metabolized by the microsomal fraction. Qualitatively, the results obtained were in agreement with those obtained from in vivo experiments.

73-0650. Torre, C. (Istituto di Anatomia Umana Normale, II Cattedra Universita di Torino, Turin, Italy). **Observations sur l'effet du DFP chez l'embryon de poulet selon le moment de l'administration. [Observations on the effect of DFP on chicken embryos as affected by the moment of administration.]** *C.R. Ass. Anat. 148: 572-575; 1970.* (5 references) (French)

The effect of single 0.5 mg doses of DFP injected into eggs between the fourth and fifteenth days of incubation was studied on chicken embryos. The DFP affected the acetylcholinesterase activity during the whole embryonic development, but it caused total death of the embryos only if it was introduced after the ninth day of incubation. The mortality of embryos treated with DFP on the sixth and seventh days of incubation was very small, and all embryos treated before the fifth day of incubation survived. Relationship between the mortality and the degree of maturity of the cholinergic system is possible.

73-0651. Raith, L. (I. Medizinische Klinik, Universitaet Muenchen, Munich, Germany). **Die medikamentoese Therapie der Nebennierenrindenueberfunktion. [Chemotherapy of adrenocortical hyperfunction.]** *Deut. Med. Wochenschr. 97(12): 475-477; 1972.* (10 references) (German)

Chemotherapy of adrenocortical hyperfunction in general and the TDE (1,1-dichloro-2-(2-chlorophenyl)-2-(4-chlorophenyl)-ethane) therapy of inoperable adrenocortical carcinoma are described. TDE has cytotoxic effects on the cells of the zona fasciculata and of the zona reticularis, but relatively slight effect on the glomerulosa cells. TDE also seems to modify the extra-adrenal metabolism of the steroids. The therapy of adrenocortical carcinoma with 3-12 g/day doses of TDE results in a considerable decline in the steroid secretion rate in about 70% of the cases and in a transient inhibition of the tumor growth or in regression in about one third of the cases. Regression of the metastases in some cases is also observed. Nausea, diarrhea, neuromuscular disorders, somnolence, and allergic skin efflorescences are common side effects of TDE therapy.

73-0652. Yamada, K.; Yamazoe, H.; Watanabe, F.; Tanabe, T.; Motoi, E.; Koshimura, S.; Murasawa, K. (Kobe Women's College of Pharmacy, Motoyama, Higashinada, Kobe, Japan). **Acute and subacute toxicity test of 2-azido-4-isopropylamino-6-methylthio-*s*-triazine(Aziprotryne, C-7019).** *Eisei Kagaku (J. Hyg. Chem.) 17(5): 310-326; 1971.*

The 48 hr acute LD50 for 2-azido-4-isopropylamino-6-methylthio-*s*-triazine (C-7019) was 2970 mg/kg in male mice and 3600 mg/kg in male rats. Sedation, ataxia, and respiratory depression were evident within 60 min of administration. After intraperitoneal administration, central excitation was observed before these symptoms and the LD50 was 265 mg/kg in male mice. Three-month subacute studies in mice and rats with doses slightly lower than 30, 50, 100, and 400 mg/kg/day in the diet revealed no abnormal behavior. Food consumption, blood, renal function, liver function, and macroscopic appearances of internal organs were normal. A slight reduction in rate of body weight gain was observed in treated rats although no dose response

was obtained. Treated animals showed no adverse histopathological changes in brain, lung, thymus, adrenal, spleen, testis, ovary, or bone marrow except infiltration of perivascular round cells in kidneys and focal cell infiltration in hepatic cells, which could be attributed to C-7019.

73-0653. Petrova-Vergieva, T.; Ivanova-Chemishanska, L. (Unified Sci. Res. Inst. Hyg. and Occup. Safety, Sofia, Bulgaria). **Kum teratogenniya efekt na tsink-etilenbisditiokarbamat (tsineb) u plukhove. [Teratogenicity of zinc ethylenebisdithiocarbamate (zineb) in rats.]** *Eksp. Med. Morfol. 10(4): 226-230; 1971.* (4 references) (Bulgarian)

Single doses of zineb perorally administered to white rats on the seventh, ninth, eleventh, or thirteenth day of pregnancy at 2000, 4000, or 8000 mg/kg (corresponding to one-fifth, two-fifths, and four-fifths of the LD50) caused a wide range of dose and day of treatment dependent teratogenic effects. Anomalies included hydrocephalus, exophthalmia, and cleft palate as well as amelia, oligodactylia, ectrodactylia, microcaudia, acaudia, and deformed tails. The most severe teratogenic effects were observed when zineb was administered on the eleventh and on the thirteenth day of pregnancy coinciding with the period of fetal organogenesis. Fetuses were most sensitive to the 4000 and 8000 mg/kg doses administered on the eleventh day of gestation; a 25% fetal mortality rate resulted, and skeletal anomalies occurred in all the surviving fetuses. The 2000 mg/kg zineb dose was the minimum dose required to cause teratogenic effects. Administration of the latter for four days between the tenth and thirteenth day of gestation produced effects similar to those caused by a single dose of 8000 mg/kg zineb administered on the eleventh day of pregnancy. Daily administration of 1000 mg/kg zineb between the second and twentieth days of pregnancy produced no fetal anomalies, indicating that zineb may be considered a low teratogenicity compound.

73-0654. Rashev, Z. (Unified Sci. Res. Inst. Hyg. and Occup. Safety, Sofia, Bulgaria). **Tsitopatichno i tsitotoksichno deystvie na nyakoy pestitsidi vurkhu embrionalni kletuchny kulturi. [Cytopathogenic and cytotoxic action of some pesticides on embryonal cell cultures.]** *Eksp. Med. Morfol. 11(1): 35-39; 1972.* (6 references) (Bulgarian)

Cytopathogenicity and cytotoxicity of Metathion (fenitrothion) and pyrocine (the Bulgarian preparation of zineb) in vivo and in vitro were studied using rat embryo fibroblast monolayer cultures. Pregnant rats were treated with one-fiftieth and one-one hundredth LD50 of Metathion or with a no-effect dose of zineb daily during the first 15 days of gestation for the in vivo experiment. Fetuses were then removed and treated to obtain fibroblasts for monolayer cultivation. In vitro studies were performed with fibroblasts, obtained likewise from intact rats by addition of 0.1 and 1.0 mg of pesticide per ml of fibroblast suspension. Mitotic studies and cytotoxicity were investigated 24 hours later upon establishment of the monolayer culture. In vivo treatment caused no differences in cell proliferation or mitotic phase distribution in comparison to controls. The total number of anomalous mitoses, including loss or irregular distribution of chromosomes along the equator of the spindle or loss of chromosomal matter during the anaphase, exceeded that of controls. The in vitro experiment indicated a considerable drop in cell proliferation rate and telophase percent and an increased number of anomalous mitoses. Cytotoxic effects, including damage of the monolayer, decreased cell staining capacity, and nuclear membrane damage, were observed in both in vivo and in vitro pesticide treated cell cultures. Cytopathogenicity and cytotoxicity of fenitrothion and zineb could not be correlated with dosages.

73-0655. Hrdina, P. D.; Singhal, R. L.; Peters, D. A. V.; Ling, G. M. (Dept. of Pharmacol., Fac. of Med., Univ. of Ottawa, Ottawa, Canada). **Comparison of the chronic effects of *p,p'*-DDT and α-chlordane on brain amines.** *Eur. J. Pharmacol. 20(1): 114-117; 1972.* (12 references)

Significant decreases in the amount of striatal ACh were observed in rats treated with 0.5 or 2.5 mg *p,p'*-DDT/100 g for 45 days, and no changes were observed with the same doses of α-chlordane. Animals receiving the higher dose of DDT showed a significant increase in 5-hydroxyindoleacetic acid but not serotonin in the brain stem, and ataxia was observed during the last few days of treatment. Neither of these amine levels was altered by α-chlordane administration. When the insecticides were given for only 20 days, no changes were observed in amine concentrations. Average colonic temperatures increased in each treated group about 1.5°C by the 45th day. Apparently, under the conditions used α-chlordane lacked the chronic effects of *p,p'*-DDT on the central nervous system.

73-0656. Nikolayev, A. I.; Ponomareva, L. A.; Geller, I. S.; Rozgon, M. I.; Garipova, F. Sh. (Tashkent Med. Inst., Tashkent, USSR). **Immunodepressivnoye deystviye nekotorykh yadokhimikatov. (Immunosuppressive action of some pesticides.)** *Farmakol. Toksikol. (Moscow) 35(3): 352-355; 1972.* (2 references) (Russian)

The immunosuppressive action of some organochlorine, organophosphorus, and urea derived pesticides was studied in chronic and acute animal tests. Intraperitoneal LD50 doses of fluometuron and Akar (chlorobenzilate) were administered to albino mice one hour or two days before immunization in acute tests. Rats, immunized or not, were fed 10%, 5%, 2%, and 1% of the LD50 of fluometuron, Akar, and monuron in acute tests. Immunized albino mice were fed 35 mg of methyl

mercaptophos and 50 mg of Phosphamide (dimethoate) on the 3rd, 7th, and 15th days of another experiment. All of the pesticides investigated had immunosuppressive action in terms of reduced number of immunocompetent cells in the spleen and reduced serum hemagglutinin titer. Urea derived compounds inhibited antibody formation during the early stages, and organochlorine pesticides did during the later stages of the immunization. Chronic pesticide poisoning evoked phasic response of the immune system. Stimulation of the antibody formation during the early stages and inhibition of the antibody formation during subsequent stages were observed.

73-0657. Ivanayevskiy, V. S.; Ardatova, A. N.; Yakusheva, O. V. (All-Union Inst. of Experimental Veterinary, Moscow, USSR). **Toksicheskoye deystviye granozana na organizm krupnogo rogatogo skota. [Toxic effect of Granosan on cattle.]** *Veterinariya (Moscow) 49(5): 81-83; 1972.* (Russian)

Four groups of calves were fed different amounts of Granosan in feed for 10-27 days in an experiment. Calves fed Granosan doses containing 17.72-24.23 mg of ethyl mercury chloride per kg body weight daily died in 12-30 days. The animals developed such symptoms as dysfunctions of the central and sympathetic nervous systems and of the cardiovascular system (depression, asthenia, loss of weight and appetite, increased pulse rate, ataxia, deafness, loss of sight, as well as hemorrhagic diathesis, inflammatory necrotic alterations of the gastrointestinal tract, and inflammatory dystrophic changes in the parenchymatous organs) in 10-23 days. Meat containing 12.9-14 mg of mercury per kg from such calves caused the death of puppies and kittens in 24-30 days.

73-0658. Hikita, H.; Ishikawa, T. (Sch. of Ophthalmol., Fac. of Med., Kitasato Univ., Mokadai, Sagamihara, Kanagawa Prefect., Japan). **[Experimental treatment of organophosphate poisoning. Inhibitory effects of GSH on corneal opacity in rats given Baytex.]** *Ganka Rinsho Iho (Jap. Rev. Clin. Ophthalmol.) 66(9): 832-834; 1972.* (14 references) (Japanese)

Baytex (fenthion) has been reported to produce corneal opacities in rats. The possibility of preventing the development of corneal opacities by administration of reduced glutathione (GSH) was investigated. Peroral administration of Baytex at 35 mg/kg/day for 14 days resulted in the development of an appreciable number of corneal opacities, but lower mortality than administration of 17.5, 70, or 140 mg/kg/day. Subcutaneous injection of GSH at 20 mg/kg/day appreciably reduced the incidence of corneal opacities in the test rats; injection of 5, 100, or 500 mg/kg was less effective. Intraocular application of GSH produced some decrease in corneal opacities, but this was not statistically significant. Peroral administration of GSH at 200 or 1000 mg/kg/day was ineffective. Glutathione administration did not forestall the marked drop in serum and erythrocyte cholinesterase levels which occurred after Baytex administration.

73-0659. Sugimoto, T. (Biological Res. Lab., Nihon Noyaku Co. Ltd., Tokyo, Japan). **[Studies on the reduction of ocular damage caused by Blasticidin-S, a fungicidal antibiotic.]** *Igaku to Seibutsugaku (Med. Biol.) 85(4): 173-176; 1972.* (7 references) (Japanese)

To reduce an ocular inflammatory injury occurring when Blasticidin-S gets into human eyes, studies were made on guinea pigs with a commercial protease as an anti-inflammatory agent mixed with Blasticidin-S. Ocular injuries by pure Blasticidin-S (actually its dodecylbenzenesulfonate) appeared as moderate blepharitis, conjunctival hyperemia, and corneal opacity when 15 μg (0.01 ml) was applied to the eyes of guinea pigs. No injuries occurred at a rate of 1 μg. One-hundredth ml of aqueous solution containing 15μg of Blasticidin-S and 100 μg of commercial protease (13 kinds) was applied to each eye of a group (10 animals) of guinea pigs; the symptoms were then observed for seven days. Two kinds of commercial protease, Pronase-P and Bromelain, reduced injuries the best: blepharitis and hyperemia became very slight, and corneal opacity did not appear. Other protease formulations showed little or no effect. The enzymic activity of Pronase-P and Bromelain was 54.9 and 29.7 (μg tyrosine/mg protease/min), but no relationship was found between the enzymic activity and the injury-reducing activity. The reducing activity of Bromelain and Pronase-P was unchanged by heating for 10 min at 100°C despite complete loss of enzymic activities. The active substance was dialyzed out of the cellophane membrane and precipitated by adding large amounts of acetone to the dialyzed out aqueous solution. Extraction, purification, and identification were carried out starting with the most active Pronase-P, using gel filtration on Sephadex 25, ion-exchange chromatography on CM-cellulose, paper chromatography, column chromatography, and IR. The active substance was finally identified as calcium acetate. Tests revealed that calcium acetate could reduce the ocular injuries due to 15 μg of Blasticidin-S by 50% at the rate of 2.5 μg; the injuries were seldom found at rates over 5 μg and the fungicidal activity of Blasticidin-S was unaffected.

73-0660. Reinbold, K. A.; Kapoor, I. P.; Childers, W. F.; Bruce, W. N.; Metcalf, R. L. (Sec. of Econ. Entomol., Ill. Natural History Survey, Urbana, IL). **Comparative uptake and biodegradability of DDT and methoxychlor by aquatic organisms.** *Ill. Natur. Hist. Surv. Bull. 30(6): 405-417; 1971.* (7 references)

An investigation of the uptake of DDT and methoxychlor from water measured methoxychlor concentrations of 0.8, 0.2, and 0.2 ppm in tilapia and DDT concentrations of 1.3, 0.7, and 6.8 ppm in other tilapia

after 3, 10, and 31 days exposure to 0.001 ppm of insecticide. A similar pattern was observed with higher levels of each insecticide and with green sunfish. The concentration factors for both fishes indicate that methoxychlor was metabolized to a much greater extent than DDT. TDE was the major DDT metabolite in tilapia while DDE was more prevalent in sunfish. Methoxychlor was degraded by both to the mono-*O*-demethylated product and the bis-*O*-demethylated metabolite. The DDT/methoxychlor ratio in daphnia after three days' exposure at each insecticide level ranged from 1.8 to 2.5, similar to that observed in fishes. Daphnia excreted methoxychlor more rapidly than DDT while the opposite was true of snails. When daphnia containing labeled DDT or methoxychlor were fed to guppies, DDT was rapidly concentrated in fish, reaching levels of 8 ppm after 20 days while methoxychlor concentrations were never above 0.17 ppm.

73-0661. Gabliks, J. (Biol. Dept., Northeastern Univ., Boston, MA 02115). **Protective action of 2,2-bis(parachlorphenyl)-1,1,1-trichloroethane against intoxication of cells by staphylococcal enterotoxin-B.** *Infec. Immunity 6(3): 364-369; 1972.* (17 references)

Both 24 and 4 hr exposure to 12.5, 25, or 50 μg/ml DDT prior to addition of staphylococcal enterotoxin-B (SEB) protected the cells of human embryonic intestine, Henle strain, against its cytotoxic effects. DDT alone did not produce any cytotoxicity detectable by microscopy. An increasingly therapeutic effect was observed when doses of 4 to 16 μg DDT/ml were added to the medium seven hr after addition of SEB with complete protection afforded at the 16 μg level. Administration of TDE (50 μg/ml) up to 9 hr after the addition of SEB also provided almost complete protection. Since cells treated with DDT for a period of 24 hr remained resistant to the toxin when DDT was removed from the medium, it appears that DDT acts on the cells rather than on the toxin itself. Toxin-induced skin erythema and necrosis in guinea pigs was prevented by intraperitoneal injection of DDT (25 mg/kg) 4 hr prior to administration of SEB.

73-0662. Davison, K. L.; Sell, J. L. (U.S. Dept. of Agr., Anim. Sci. Res. Div., Agr. Res. Serv., Metabolism and Radiation Res. Lab., Fargo, ND 58102). **Dieldrin and *p,p'*-DDT effects on some microsomal enzymes of livers of chickens and mallard ducks.** *J. Agr. Food Chem. 20(6): 1198-1205; 1972.* (15 references)

Two experiments were conducted to determine effects of dieldrin and *p,p'*-DDT on some mixed-function oxidases of hepatic microsomes of White Leghorn chickens and mallard ducks. Aniline hydroxylase and aminopyrine *N*-demethylase activities in control birds were lower in ducks than in chickens, whereas cytochrome P450 concentration and estradiol metabolism in microsomes were about equal in both species. Dieldrin at levels of 10 and 20 μg/g of diet and DDT at 100 and 200 μg/g of diet increased cytochrome P450 concentration and estradiol metabolism in microsomes of both species, but they increased these parameters more in ducks than in chickens. DDT decreased aniline hydroxylase activity in chicken microsomes and increased aniline hydroxylase activity in duck microsomes, but did not affect aminopyrine *N*-demethylase activity of either species. Dieldrin did not affect aniline hydroxylase activity in chickens or ducks and did not affect aminopyrine *N*-demethylase activity in ducks, but it probably increased *N*-demethylase activity in chickens. (Author abstract reprinted by permission of the American Chemical Society)

73-0663. Laanio, T. L.; Dupuis, G.; Esser, H. O.* (Agrochemicals Division, Ciba-Geigy Ltd., Basel, Switzerland). **Fate of ^{14}C-labeled diazinon in rice, paddy soil, and pea plants.** *J. Agr. Food Chem. 20(6): 1213-1219; 1972.* (39 references)

[2-^{14}C]Diazinon [*O,O*-diethyl *O*-(2-isopropyl-4-methylpyrimidin-6-yl) phosphorothioate] was rapidly absorbed by and translocated in rice plants. Loss of insecticide, of about 50% within 9 days, was due to volatilization from the paddy water and transpiration from the leaves. Less than 10% of the radioactivity remaining in the plants after 9 days was the parent compound. The metabolite fraction consisted of 2-isopropyl-4-methyl-6-hydroxypyrimidine (G 27550), 2-(1′-hydroxy-1′-methyl)ethyl-4-methyl-6-hydroxypyrimidine (GS 31144), the latter partly as a glucoside, and a small fraction of polar metabolites. The same metabolites and traces of diazoxon were found after stem injection of the insecticide. Hydroxydiazinon [*O,O*-diethyl *O*-[2-(1′-hydroxy- 1′-methyl)-ethyl-4-methylpyrimidin-6-yl] phosphorothioate] if present represented only a minor metabolite. G 27550 and GS 31144 were also the main metabolites of paddy soil. Cleavage of the pyrimidine ring with the evolution of $^{14}CO_2$ proceeded at a low rate in rice plants and paddy soil. The degradative pathways found in rice also occurred in pea plants. (Author abstract reprinted by permission of the American Chemical Society)

73-0664. Adler, I. L., Haines, L. D.; Wargo, Jr., J. P. (Research Laboratories, Rohm & Haas Company, Bristol, PA 19007). **Studies with dairy cows and laying hens fed alfalfa containing field-aged residues due to 3,5-dichloro-*N*-(1,1-dimethyl-2-propyl)benzamide.** *J. Agr. Food Chem. 20(6): 1233-1235; 1972.* (4 references)

Groups of dairy cows and laying hens were fed specially prepared diets containing alfalfa with field-aged residues due to the herbicide, 3,5-dichloro-*N*(1,1-dimethyl-2-propynyl)benzamide(Kerb). Residues greater than 0.01 ppm were found only in milk from cows fed 7.5 ppm of herbicide residues and in eggs from chickens

fed 1.8 ppm of herbicide residues. No evidence for a continual accumulation of residues was observed in either eggs or milk. Detectable residues were found only in cow liver and kidney, and hen liver, kidney, gizzard, and fat. (Author abstract reprinted by permission of the American Chemical Society)

73-0665. Bandal, S. K.; Casida, J. E.* (Div. of Entomol., Univ. of California, Berkeley, CA 94720). **Metabolism and photoalteration of 2-*sec*-butyl-4,6-dinitrophenol (DNBP herbicide) and its isopropyl carbonate derivative (dinobuton acaricide).** *J. Agr. Food Chem. 20(6): 1235-1245; 1972.* (27 references)

Metabolism of 2-*sec*-butyl-4,6-dinitrophenyl isopropyl carbonate (dinobuton) in mice and rats involves rapid hydrolysis to form 2-*sec*-butyl-4,6-dinitrophenol (DNBP) which, in turn, undergoes oxidation of either of the two methyl groups on the *sec*-butyl side chain, conjugation of the phenolic products, formation of many uncharacterized metabolites, and, in rats only, reduction of either of the two nitro groups and acetylation of the metabolically formed *p*-amino group. The most active esterases for dinobuton hydrolysis are present in liver and blood, but those in blood possibly are most important in the release of the actual toxicant, DNBP, and in determining the relative toxicity of dinobuton to various mammalian species. Carbaryl protects the rat from poisoning by dinobuton, probably by inhibiting its hydrolysis to DNBP. Microsomal enzymes of rat liver and housefly abdomens hydrolyze dinobuton and reduce the *o*-nitro group of DNBP. Dinobuton and DNBP are not highly systemic in bean plants, but they are metabolized in or photodecomposed on plants to yield many products by hydrolysis, reduction, and other types of reactions. (Author abstract reprinted by permission of the American Chemical Society).

73-0666. Gibson, J. R.; Ivie, G. W.; Dorough, H. W.* (Dept. of Entomol., Univ. of Kentucky, Lexington, KY 40506). **Fate of mirex and its major photodecomposition product in rats.** *J. Agr. Food Chem. 20(6): 1246-1248; 1972.* (13 references)

Rats receiving single oral doses of radiolabeled mirex (0.5, μCi, 0.2 mg/kg) eliminated about 18% of the administered radiocarbon during a 7-day period. Of the total quantity eliminated, 85% was excreted in the feces within 48 hr after administration. Only trace amounts of radioactivity were detected in the urine. Essentially all of the excreted radioactivity was as unmetabolized mirex. Prefeeding rats with unlabeled mirex resulted in a slight decrease in intestinal absorption of a single oral dose of the ^{14}C material. A major mirex photoproduct was prepared and its chemical nature and fate in rats were evaluated. This product exhibited almost identical behavior to mirex after oral administration to rats, in that it was not metabolized by the animal to any appreciable extent. Both mirex and its photoproduct showed strong lipophilic tendencies, with concentrations of mirex equivalents in fat 7 days after treatment being 0.9 and 1.1 ppm, respectively. Mirex levels in fat did not decline below the 7-day level when the experimental period was extended to 28 days. (Author abstract reprinted by permission of the American Chemical Society)

73-0667. Kellner, H. M.; Drepper, K; Kedenburg, C. P.; Christ, O. E. (Farbwerke Hoechst A.G., vormals Meister Lucius & Bruening, Frankfurt (Main), Hoechst, Germany). **Absorption, distribution, and excretion of the ^{14}C- labeled acaricide Hoe 2910 following oral application to milk-producing ruminants and dermal application to a calf.** *J. Agr. Food Chem. 20(6): 1249-1252; 1972.* (3 references)

Orally as well as dermally applied Hoe 2910-^{14}C (Benoxaphos-^{14}C) was absorbed; the percutaneous absorption had only a subordinate significance compared to the enteral. The elimination proceeded rapidly; the half-lives for urine, feces, and milk were on the average 15 ± 2.4 hr for the sheep and 18 ± 4 hr for the cow. The amounts eliminated with milk were small, and for the sheep were less than 0.2% and for the cow less than 1% of the administered dosage. The residue in the organism of the sheep was only 0.5% of the administered radioactivity four days after administration and for the cow was only about 1% five days after administration. Of those organs and tissues intended for human consumption only the liver and (of the sheep) the retroperitoneal fat contained concentrations of 0.1 μg per gram at those times; the content of the muscles was only 0.01 μg per gram. The concentrations in the calf that was killed 13 days after dermal application were considerably lower. (Author abstract reprinted by permission of the American Chemical Society)

73-0668. Feland, B.; Smith, J. T.* (Dept. of Nutrition, Univ. of Tennessee, Knoxville, TN 37916). **Malathion intoxication and mitochondrial damage.** *J. Agr. Food Chem. 20(6): 1274-1275; 1972.* (13 references)

There was a decrease in the hexosamine content of intact liver mitochondria but not nuclei from malathion fed rats. The decreased hexosamine content coupled with decreased $^{32}SO_4^{2-}$ uptake indicated loss of mucopolysaccharide from the mitochondria. Extraction procedures indicated that in addition to mucopolysaccharide loss there was also a loss in nitrogen from the mitochondrial membrane. A decrease in swelling confirmed the apparent loss in mitochondrial integrity following malathion feeding. (Author abstract reprinted by permission of the American Chemical Society)

73-0669. Parish, R. C.; Trei, J. E. (Smith Kline and French, Applebrook Animal Health Res. Cent., West Chester, PA 19830). **Effect of DDT on rumen fermenta-**

tion. *J. Agr. Food Chem. 20(6): 1284-1286; 1972.* (20 references)

The effect of DDT, TDE, other DDT metabolites and analogs, chlordane, dieldrin, aldrin, and dichlorvos on in vitro fermentation and methane production by rumen microbes was studied. DDT inhibited methane production only at very high levels (240 ppm) in contrast to chloral hydrate, which is a potent methane inhibitor at 12 ppm. DDT and other pesticides studied had no effect on fermentation gases or volatile fatty acid metabolites at 120 ppm. (Author abstract reprinted by permission of the American Chemical Society)

73-0670. Salvador, R. L.; Saucier, M.; Simon, D.; Goyer, R. (Fac. of Pharm., Univ. of Montreal, Montreal, Canada). **Acetylcholinesterase reactivators Pyridyl and anilyl trifluoromethyl ketoximes.** *J. Med. Chem. 15(6): 646-650; 1972.* (38 references)

Several new ketoxime isomers of pyridine and *N,N*-dimethylaniline were synthesized and evaluated as reactivators of AChE inhibited by Sarin. In vitro, the 3-pyridinium trifluoromethyl ketoxime methochloride was the most active of the new series and 7 times less active than 2-PAM. The pyridinium trifluoromethyl ketoximes were as good as or better than their corresponding aldoximes and most of the pyridinium ketoximes tested as reactivators. All of the compounds which looked promising in vitro had no antidotal effect against poisoning with paraoxon or Sarin in mice. However, protective activity against these poisons was observed. One compound was an excellent protecting agent against paraoxon at a dose of 348 μmoles/kg, surpassing the combination of 17.5 μmoles/kg 2-PAM-Cl and 50 μmoles/kg caramiphen. The compound in combination with caramiphen offered good protection against Sarin while 2-PAM-Cl and caramiphen were ineffective. The quaternary salts of the bases were almost inactive in vivo.

73-0671. Sharma, R. D.; Gautam, O. P. (Dept. of Vet. Med., College of Vet. Med., Hissar, India). **Experimental endrin poisoning in calves.** *J. Res. Punjab Agr. Univ. 8(3): 394-403; 1971.* (13 references)

Experimental poisoning was induced in four calves by exposing them to endrin-sprayed grass and in nine calves by drenching with endrin. The most prominent symptom, observed within 24 hr, was nervous excitement accompanied by convulsions and an accelerated respiratory rate. A rise in temperature, profuse salivation, eye and nasal discharges, and sensitivity in the lumbar region were also noted. Calves which were drenched with 1 mg endrin/lb on the first day and 1 mg/lb after 48 hr did not exhibit nervous excitement. In these animals depression was observed followed by unconsciousness, emaciation, dehydration, and death. Changes in blood composition and marked albuminuria were observed during poisoning. Treatment with chloral hydrate, calcium gluconate, magnesium sulfate, and treacle gave good results. Those animals which did not respond to treatment showed marked gelatinization of kidney fat, along with congestion and degenerative changes of kidneys and lesions in the liver, lungs, and gut. Endrin was determined qualitatively in brain and liver. The results indicate that normal application of endrin on crops is sufficient to cause poisoning if animals have access to the sprayed fodder.

73-0672. Van Velzen, A. C.; Stiles, W. B.; Stickel, L. F. (Patuxent Wildlife Res. Cent., BSFW, Laurel, MD). **Lethal mobilization of DDT by cowbirds.** *J. Wildl. Manage. 36(3): 733-739; 1972.* (21 references)

The effect of mobilization of DDT during reduced food intake was determined experimentally in cowbirds. Of the 20 birds fed 100 ppm DDT in the diet for 13 days, untreated diet for two days, and then reduced, untreated diet, seven died during four days of food restriction. Results with control groups indicated that weight loss or dosage alone was not suffuicient to produce mortality. During food restriction, weight loss averaged 19% among untreated birds, 18% among DDT-dosed birds, and 21% among DDT-dosed birds that died. Characteristic signs of DDT poisoning were observed on necropsy of birds that died. Brain DDT residues, far below the lethal range before food restriction, increased 17.4 fold in birds that died during restriction. Brain TDE and DDE also increased in these birds. Birds that died had 16% less total DDT than those sacrificed before weight loss, body weights were 21% lower and lipid weights were 81% lower. Mortality also occurred among treated birds fed untreated diets for four months and then reduced, untreated diets.

73-0673. Fikes, M. H.; Tubb, R. A. (Ohio Coop. Fish. Unit, Ohio State Univ., Columbus, OH). **Dieldrin uptake in the three-ridge naiad.** *J. Wildl. Manage. 36(3): 802-809; 1972.* (10 references)

Uptake, retention, and release of dieldrin by naiad mollusks was similar for concentrations of 20 ppb and 20 ppt in a continuous flow system. Rapid accumulation in the gills occurred within the first two weeks of exposure, followed by stabilization at 17 ppm and 55 ppb, respectively, and then after no further addition of dieldrin, rapid release. Release rate of dieldrin by naiads held at 20 ppb was one sixth that of the uptake rate, and those exposed to 20 ppt released dieldrin at one fourth the uptake rate. The results indicate that naiads living in the water could be used for detecting low levels of dieldrin or, if suspended for only brief periods, to detect larger quantities.

73-0674. Gajdos, A. (Lab. de Rech. Biochim., Univ. Paris, Paris, France). **Le cytochrome P-450. Detoxication par les microsomes hepatiques. [Cytochrome P-450.**

Detoxication by hepatic microsomes.] *Nouv. Presse Med. 1(23): 1587-1592; 1972.* (4 references) (French)

Studies concerning the hepatic microsomal system which oxidizes endogenous and exogenous substances are reviewed. Cytochrome P-450, cytochrome P-450 reductase, and NADPH are the basic components of the hepatic oxidizing system. Exogenous substances such as pesticides combine with cytochrome P-450 during their oxidation for which the hydrogen is supplied by NADPH. Increased cytochrome P-450 and cytochrome P-450 reductase levels during detoxication are observed. The hepatic microsomal oxidizing system has highly adaptative, inducing properties for the detoxication of a great variety of substances. Sex-related differences in the hepatic microsomal activity in certain animals such as rats are possibly due to the effect of androgenic hormones.

73-0675. Eyer, H. (Max-von-Pettenkofer-Inst. fuer. Hyg. und. Med. Mikrobiol., Muenchen Univ., Munich, Germany). **DDT als "Insektenpulver" beim Menschen. [DDT as "insect powder" for human use.]** *Muenchen. Med. Wochenschr. 114: 409-410; 1972.* (German)

In response to a reader's question, the nearly general ban on the use of DDT as an insecticide is attributed to its high persistence and accumulation in the organism and to resistance developed by several insects against DDT. These insects such as lice and gnats produce an enzyme capable of converting DDT into a practically ineffective metabolite. Also, there are synthetic and natural substances which are just as effective as DDT against crawling insects without having oral toxicity for humans and animals.

73-0676. Bock, R.; Freitag, K-D. (Inst. fuer Anorgan. Chem. und Kernchem., Univ. Mainz, Mainz, Germany). **Abbau von Triphenylzinnchlorid ("Fentinchlorid") auf der Pflanze. [Degradation of triphenyltin chloride ("fentin chloride") on plants.]** *Naturwissenschaften 59(4): 165-166; 1972.* (German)

Triphenyltin compounds on plants are broken down to diphenyl- and monophenyltin compounds as well as inorganic tin (IV), according to studies performed with triphenyltin chloride applied to sugar beets in a greenhouse. High accumulations of diphenyl- and monophenyltin compounds compared to the starting material are not possible since the degradation rate increases with decreasing number of phenyl groups.

73-0677. Van Daalen, J. J.; Meltzer, J.; Mulder, R.*; Wellinga, K. (Res. Lab. N. V. Philips-Duphar, 's-Graveland, 1323, The Netherlands). **A selective insecticide with a novel mode of action.** *Naturwissenschaften 59(7): 312-313; 1972.*

A compound related to dichlobenil, 1-(2,6-dichlorobenzoyl)-3-(3,4-dichlorophenyl)-urea, had low toxicity to warm-blooded animals and plants and useful insecticidal properties. The acute oral LD50 to mice was >3160 mg/kg and the acute intraperitoneal LD50 was >1000 mg/kg. No animals died in these experiments. A pre-emergence application of the compound at a rate of 10 kg/ha did not inhibit germination of seeds, and leaves of the French bean, tomato, sugar beet, oats, and weeds were unaffected by spraying with a 1% acetonic solution. Since the mode of action in insects appeared to be connected with moulting, specificity toward arthropods is expected.

73-0678. Hochstrate, C.; Oberdisse, E. (Pharmakologisches Inst., Freie Universitaet Berlin, Berlin, Germany). **Biochemische Veraenderung im Serum der Ratte nach kombinierter Applikation lebervergroessernder und lebertoxischer Pharmaka. [Biochemical change in rat serum following combined application of hepatomegalia-inducing and hepatotoxic drugs.]** *Naunyn Schmiedebergs Arch. Pharmakol. Exp. Pathol. 266: 357-358; 1970.* (1 reference) (German)

The serum enzyme activity was studied in rats treated with an i.v. 14 mg/kg dose of praseodymium nitrate or an intraperitoneal 0.7 mg/kg dose of carbon tetrachloride after pretreatment with three 100 mg/kg oral doses of phenobarbital or with an 200 mg/kg oral dose of alpha-hexachlorocyclohexane (α-BHC). Change in the serum enzyme activity occurred only when induction occurred at the moment of the hepatic lesion. Both increased effect of CCl_4 and enzymatic activity were observed after 24 hr. While CCl_4 alone was not lethal, about 30% of all animals died due to the combined effect of CCl_4 and phenobarbital during the first 24 hr. Combinations of praseodymium nitrate with alpha-BHC or phenobarbital were not lethal either. The hepatomegalia-inducing effect of praseodymium nitrate was reduced by alpha-BHC. The findings suggest increased synthesis of phosphoric acid-containing metabolites due to pretreatment with inducing substances which results in the maintenance of the physiological equilibrium.

73-0679. Ogata, S. (Dept. Ophthalmol., Ichikawa Hospital, Tokyo Dental College, Ichikawa, Japan). **[Effects of organophosphorus pesticides on beagles in chronic toxicity studies (Report 1).]** *Nippon Ganka Gakkai Zasshi (J. Jap. Ophthalmol. Soc.) 76(9): 1143-1150; 1972.* (8 references) (Japanese)

Fenitrothion and fenthion were separately administered orally in capsules to a group (2 animals per group) of female beagles at a daily dose of 2 mg/kg for 379 days. Findings of the examination indicated blood ChE activity began to decrease after two weeks and erythrocyte ChE took a lower and steady value (about 40% of control) two months after the beginning of administration. Plasma ChE activity also clearly decreased, but fluctuated. GOT, GPT, alkaline phosphatase, and urea nitrogen did not differ from controls. Except for some

increase in ocular secretions and salivation and diarrhea in some animals, no anomalies were observed in general health conditions. Macroscopically, no conspicuous miosis, mydriasis, unevenness between eyes, or abnormal light reflex could be observed. Ophthalmic pressures of all the animals were constant during the experiment; no conspicuous tendency towards myopia or hypermetropia appeared. Although no large change appeared in the corneal curvature radius along the horizontal axis, a slight decrease appeared in that along the perpendicular axis. No change was observed along the eye fundus and no histological change was seen in the eyes. Except for a decrease of ChE activity in liver and kidney, no histochemical anomaly could be detected. Further studies will determine at what dosage anomalies occur.

73-0680. Takase, I.; Tsuda, H. (Agr. Chem. Inst., Nihon Tokushu Noyaku Seizo Co., Ltd., Hinoshi, Tokyo, Japan). **[Studies on the gas effect of disulfoton.]** *Nippon Oyo Dobutsu Konchu Gakkai-shi (Jap. J. Appl. Entomol. Zool.) 16(1): 32-39; 1972.* (15 references) (Japanese)

The effects and metabolism of disulfoton, applied as a gas, were investigated using bean plants grown in sealed containers. Disulfoton and its metabolites were determined in extracts of various plant parts. After exposure to disulfoton vapor for 24 hr, the plants contained 10.0 ppm of disulfoton and its oxidative metabolites: 70% was unchanged disulfoton, 13% disulfoton sulfoxide, 14% disulfoton sulfone, and 3% oxygen analogs of disulfoton. Disulfoton applied to soil was metabolized by plant and soil to the sulfoxide and sulfone derivatives and oxygen analogs of disulfoton. In the case of direct application to soil, only a small fraction of the activity present was in the form of disulfoton. Disulfoton, even when applied to the soil, may exert a fumigant effect due to its high volatility and vaporization.

73-0681. Sakaguchi, H. (Fish Experiment Station, Nakasuki, Akashi, Hyogo, Japan). **[Effects of agricultural chemicals on fish. I. Changes in chemical components in serum and liver of carp exposed to organophosphate compounds.]** *Nippon Suisan Gakkai-shi. (Bull. Jap. Soc. Sci. Fish.) 38(6): 555-560; 1972.* (26 references) (Japanese)

Physiological changes were observed in carp, *Cyprinus carpio,* exposed to the organophosphate compounds dipterex (trichlorfon), malathion, and dichlorvos at 2-10 ppm. Fish were maintained in 5-l. vinyl tanks of water containing the chemicals. The SGOT and SGPT levels were increased and serum cholinesterase decreased in all groups of treated fish. Serum amylase was unchanged. Serum glucose was markedly elevated and serum triglycerides and total cholesterol reduced while total serum protein was not significantly altered. Liver glycogen content was reduced by 25% in fish exposed to dichlorvos at all concentrations tested. Liver lipid contents showed variable changes.

73-0682. Oshiba, K. (Sch. of Hyg. and Pub. Health, Fac. of Med., Osaka Municipal Univ., Osaka, Japan). **[Experimental studies on the fate of β-and γ-BHC in vivo following daily administration.]** *Osaka Shiritsu Daigaku Igaku Zasshi (J. Osaka City Med. Cent.) 21(1-3): 1-9; 1972.* (44 references) (Japanese)

Several groups of female Sprague Dawley rats were fed a diet low in proteins and fats corresponding to the common Japanese diet and containing respectively 1, 10, and 100 ppm of γ-BHC (lindane) and 10 ppm of β-BHC for different periods (1 to 56 days). The rats were sacrificed at predetermined intervals to determine the distribution of BHC, the deposition of BHC-isomers, and their variation with the lapse of time. Studies were also made on the absorption and excretion of ^{14}C-labeled γ-BHC. Only the 100 ppm group showed a marked decrease of body weight after 42 days of feeding; the slow decrease in food intake began on the 35th day of feeding and with a slight increase of the weight of the liver. BHC was found in all organs and tissues examined (liver, kidney, brain, adipose tissue, and blood), and was especially high in adipose tissue. As a rule, the deposition of β-BHC was higher than that of γ-BHC in most of the organs and tissues, but the reverse was true in the blood. The concentration of γ-BHC in the organs and tissues increased rapidly and plateaued within 3 to 7 days in the 100 ppm group at 55 μg/g tissue lipid in liver and 163 μg/g in adipose tissue; the maximum deposition was seen in 1 and 10 ppm groups on the 21st and 14th day, respectively. The deposition of β-isomer in white adipose tissue continued to increase throughout the 56 days of feeding. Conditions known to accelerate the lipid metabolism markedly reduced γ-BHC already deposited in organs and tissues; the effect in reducing β-BHC was slight. When the animals were reduced to a starved condition, β-BHC was mobilized from adipose tissue by an enhanced lipid metabolism and tended to be deposited in other organs and tissues. A protein-rich diet was effective in reducing BHC, especially β-isomer. It was found that the effects were roughly proportional to the protein content of the diet. The results of radio-tracer studies indicated that about 80% of β-BHC ingested was absorbed from the digestive tract. The BHC once deposited in the organs and tissues was excreted mostly in urine and partly in feces. It is concluded from the above findings that the transfer of BHC among different organs and tissues takes place in close relation to the lipid metabolism and that dietary protein plays an important role in the detoxication of BHC in the liver.

73-0683. Sosnierz, M.; Szczurek. Z.; Knapek, R.; Kolodziejczyk, A. (Inst. of Organic Industry, Pszczyna, Poland). **Badania morfotyczne nad skrocona toksycznoscia przewlekla kamfochloru. [Morphologic studies**

on short-term subacute toxicity of Kamfochlor.] *Patol. Pol. 23(2): 199-202; 1972.* (5 references) (Polish)

Kamfochlor (toxaphene) was administered orally as a 25% olive oil solution in 0.07, 0.22, 0.66, 2.0, and 6.0 mg/kg/day dose to groups of Wistar rats (male and female) for 90 days. Examination of liver, spleen, kidneys, lungs, and testes revealed pathomorphologic changes in animals receiving high dosages (2 and 6 mg/kg) of the compound. These included circulation disorders (mainly in lungs and spleen) and degenerative lesions (liver and kidneys). Impaired circulation caused congestion and damaged blood vessel walls resulting in hyperemia, extravasation, and hemorrhage. The compound was also cytotoxic, progressively damaging and diminishing the activity of cell organelles and leading to death of cells. In the spleen a reduction of Malpighian follicles was noted suggesting an inhibition of lymphopoiesis. Lesions in the testes indicated a harmful effect on spermatogenesis. There was no difference in severity of changes in male as compared to female rats, but individual sensitivity to the compound differed among animals.

73-0684. Hammann, I. (Pflanzenschutz Anwendungstech. Abt., Biol. Forschung, Farbenfabriken Bayer AG, Leverkusen, Germany). **Tamaron, a new insecticide and acaricide.** *Pflanzenschutz-Nachr. 23(2): 133-143; 1970.* (6 references)

O,S-Dimethyl-phosphoramidothiolate, the active ingredient of Tamaron (Monitor), a new organophosphate insecticide, had an acute oral LD50 to rats and mice of 30 mg/kg. Dermal LD50 in rats was about 50 mg/kg when the active ingredient was not removed from the skin after a period of time. A 4-hr inhalation exposure to male rats yielded an LC50 of 162 mg/kg. In subchronic toxicity studies no-effect levels of 2 and 15 ppm in the diet were observed for rats and dogs, respectively. The LC50 values for four species of fish tested at 96-hr exposure ranged from 10 to 100 ppm. Good plant tolerance to the compound has been recorded both in the laboratory and in the field. Tamaron provides simultaneous control of practically all economically important pests of a crop, eradicates many hard-to-kill insects as well as those with concealed habits, and is effective against various resistant species.

73-0685. Homeyer, B. (Pflanzenschutz Anwendungstech. Abt., Biol. Forschung, Farbenfabriken Bayer AG, Leverkusen, Germany). **Present state of soil insect pest control.** *Pflanzenschutz-Nachr. 23(3): 224-230; 1972.* (7 references)

Chlorinated hydrocarbons replaced fumigants as soil insecticides because of easier application and better control. These are being replaced at present because of the development of resistance to them among insects and their persistence in the environment. Fensulfothion and phoxim are two new organophosphates which look promising as soil insecticides. Both compounds control a wide range of insects formerly controlled by the chlorinated hydrocarbons. They were found to have better penetration in soil than aldrin and to persist under laboratory conditions for about 4 mo. and under field conditions for 2.5 to 3 mo. Phoxim is much less toxic to mammals, having an oral LD50 in male rats of 2066 mg/kg compared to an oral LD50 of between 4 and 10 mg/kg for fensulfothion.

73-0686. Loeser, E.; Kimmerle, G. (Institut fuer Toxikol., Farbenfabriken Bayer AG, Wuppertal-Elberfeld, Germany). **Acute and subchronic toxicity of Nemacur active ingredient.** *Pflanzenschutz-Nachr. 24(1): 69-113; 1971.* (27 references)

Extensive acute and subacute toxicity studies of phenamiphos (Nemacur) indicated that poisoning symptoms were similar to those produced by other organophosphates. The nematocide was relatively toxic in acute doses to mice, rats, rabbits, cats, dogs, and hens. Oral LD50s ranged from 5.31 mg/kg (hens) to 75 to 100 mg/kg (guinea pigs). Dermal absorption was very low while absorption through the lungs was high. No damaging effects were observed on skin and mucous membranes in rabbits. Symptoms of cholinergic poisoning appeared quickly and disappeared within 24 hr. Atropine sulfate, PAM, and toxogonin (obidoxime) were very effective as antidotes to acute poisonings in rats and hens. Liver and kidney functions were not affected in dogs receiving a single oral dose of 3.0 mg/kg or rabbits receiving a dose of 2.5 mg/kg orally. In vitro I50 values for ChE were 5.1×10^{-5} moles in serum, 6.3×10^{-4} moles in erythrocytes, and 2.1×10^{-4} moles in brain. The greatest inhibition of ChE in vivo occurred three hours after dosing in rats. No cumulative effects were noted in 2 mo. studies in rats given daily oral doses up to 2.5 mg/kg or intraperitoneal doses up to 1.5 mg/kg. Hematology and urinalysis showed no pathological changes in rats treated with up to 32 ppm for three months and no organ damage was observed. Levels of 4 ppm depressed plasma ChE, and levels of 16 ppm also depressed erythrocyte ChE. In the three month feeding experiment in dogs 2 ppm in the diet depressed plasma ChE and 5 ppm inhibited both enzymes. No other toxic effects were observed. A dietary concentration of up to 30 ppm did not alter fertility, lactation performance, weight of young, or survival rates.

73-0687. Senges, J.; Ruedel, R. (Med. Poliklinik and Physiol. Inst., Univ. Heidelberg, Heidelberg, Germany). **Experimental myotonia in mammalian skeletal muscle: changes in contractile properties.** *Pfluegers Arch. 331: 315-323; 1972.* (16 references)

An increase of contraction amplitude and prolongation of relaxation in curarized isolated diaphragm, resembling changes occurring in clinical myotonia, were produced by 2.5m*M* 2,4-D application. The peak ampli-

tude of single contractions increased up to 6.2 fold and the duration of relaxation was prolonged 10 to 20 fold. Not all fibers were affected by 2,4-D at the same rate. A brief pulse train elicited a stronger response than a single pulse. The peak of contraction was potentiated further and relaxation returned to nearly normal duration when a conditioning stimulus was applied 0.5 to 4 seconds before the test stimulus.

73-0688. Ruedel, R.; Senges, J. (Physiol. Inst. and Med. Poliklinik, Univ. Heidelberg, Heidelberg, Germany). **Experimental myotonia in mammalian skeletal muscle: changes in membrane properties.** *Pfluegers Arch. 331: 324-334; 1972.* (33 references)

A 2- to 2.5- fold increase in specific membrane resistance in rat diaphragms was observed after bathing the tissue in 2.5 m*M* 2,4-D or treating the animal with 20,25-diazocholesterol. The effect was noted about 10 min. after addition of 2,4-D. No changes were observed in membrane resting potential and capacitance. When Ca was omitted from the bath the mean resting potential again did not change, and the membrane resistance increased by a factor of 1.8. Experiments in Ca-free solution containing 2,4-D did not succeed because of repetitive firing of the fibers following impalement.

73-0689. Chippendale, T. J.; Zawolkow, G. A.; Russell, R. W.; Overstreet, D. H. (Univ. of California, Irvine, CA). **Tolerance to low acetylcholinesterase levels: modification of behavior without acute behavioral change.** *Psychopharmacologia (Berlin) 26(2): 127-139; 1972.* (20 references)

Daily administration of peanut oil or 0.2 mg/kg DFP in peanut oil to rats did not affect drinking behavior. After a latency of 6 days, water intake decreased markedly and then returned to normal with doses of 0.4 and 0.5 mg/kg. Drinking decreased to 0 in 4 days when the dose of DFP was 1.0 mg/kg. The latency period increased inversely with dose, as did the peak effect time. The therapeutic index, calculated to be 1.87, indicates a narrow margin of safety for DFP. Brain ChE levels decreased to an asymptote at approximately 30% of normal at different rates depending on the dose. Behavioral decrements induced by scopolamine were more severe in DFP tolerant animals than in controls. Methyl scopolamine was less active and no differences were observed between treatment groups. Challenges with α-methyl-*p*-tryosine did not affect drinking behavior.

73-0690. Hannan, P. J.; Patouillet, C. (Marine Biol. and Biochem. Branch, Ocean Sciences Div., Nav. Res. Lab., Washington, DC) **Effects of pollutants on growth of algae.** *Rep. NRL Nav. Res. Lab. Progr. 1-8; Feb. 1972.* (10 references)

A variety of pollutants were tested for effects on the growth rates of several freshwater and marine algae. Nutrients were provided in sufficient concentrations to sustain growth for three days, but not enough to negate a toxic effect. Mercury was the most toxic of the pollutants tested, and mercuric chloride had greater toxicity based on 0.1 ppm mercury than dimethylmercury. DDT had little effect on the growth rates of the algae used, probably because of its low solubility.

73-0691. Yamagishi, T.; Takeba, K.; Fujimoto, C.; Morimoto, K.; Haruta, M. (Section of Milk and Meats Hyg., Dept. of Sciences for Living, Tokyo Metropolitan Pub. Health Res. Lab., Tokyo, Japan). **[On the effects of β-BHC on the fetus of mouse (IV).]** *Rinsho Eiyo (J. Clin. Nutr.) 41(5): 599-604; 1972.* (13 references) (Japanese)

The effects of β-BHC on fetal growth were studied. β-BHC was administered s.c. and p.o. to pregnant mice of ICR-JCL strain and the fetuses removed on the eighteenth day of gestation. Mice were injected subcutaneously in the back with β-BHC dissolved in olive oil at doses of 5, 50, and 100 μg per gram of body weight once a day for five successive days from days two to six or seven to eleven. Percentage of anomalies such as immature fetuses, early death, and later death was 23.8, 35.6, and 47.3, respectively as compared to 23.6 of control mice injected in early stages of pregnancy. Fetal anomalies were 30.0, 24.6, and 21.3% respectively, compared to 20.0 of control mice injected in the middle stage of pregnancy. Another group of females was pretreated with a diet containing 50 ppm (5 mg/kg) of β-BHC for one month before mating and further administered the standard doses p.o. on days six and seven of gestation. The percentage of anomalous fetuses was 11.3, 24.0, and 25.8, respectively, as compared to 8% of controls. The deposition of β-BHC in the body of mother and fetus was correlated with the amount of β-BHC administered: in the livers of dams fed only the contaminated diet, the residue was 22 ppm; in those given 50 μg, 83 ppm; and in those given 100 μg, 118 ppm; amounts in the liver of fetus were 4, 10, and 22 ppm. The translocation of β-BHC from dam to fetus was about one-fifth to one-eighth.

73-0692. Janda, J. (Res. Inst. of Forestry and Game Management, Zbraslav, Czechoslovakia). **On the toxicity of DNOC to pheasants, partridges and hares.** *Sci. Agr. Bohemoslov. 2(4): 301-312; 1970.* (13 references)

Rafex 35 (35% DNOC) had an oral LD50 of 24 mg/kg in pheasants and partridges and 70.8 mg/kg in hare. Heavy breathing and asphyctic spasms followed by apparent stiffness of muscles characterized intoxication; signs were observed at 10 to 20 min in the birds and 2 hr in the hare. The course of intoxication in birds that survived was relatively short, and feed intake returned to normal 2 days after dosing. Repeated doses acted rapidly

and if recovery occurred it was within a short period of time. Rafex 35 showed a partial repellent effect in pheasants, but the effect was not high enough to make them avoid consuming contaminated food.

73-0693. Pujman, V.; Janda, J.; Knobloch, E.; Pokorna-Hanusova, D.; Cernochova, S.; Likovsky, Z.; Roubal, Z.; Nemec, Z.; Boseova, M.; Pav, J. (Res. Inst. of Pharm. and Biochem., Prague, Czechoslovakia). **Effect of DDT on pheasants.** *Sci. Agr. Bohemoslov. 2(4): 313-325; 1970.* (10 references)

A level of 0.02% of DDT in the diet for 14 days had no apparent effect on the behavior, food intake, or weight gain of pheasants. Livers of the experimental birds were markedly but not significantly enlarged, and weights of ovaries were lowered. The liver vitamin A level was lower in experimental birds at least up to the 16th day after removal of treated feed. Hematological examination revealed the occurrence of young granulocytes in peripheral blood and a significant reduction in the absolute number of lymphocytes. Levels of calcium and blood plasma were not affected. Red blood cell formation in bone marrow appeared to be activated 48 hr after removal of treated diet.

73-0694. Oshiba, K.; Kawakita, H. (Osaka City Inst. of Hyg., Osaka, Japan). **[Interaction between toxicant and nutrition II. Relationship between concentrations of γ-BHC in diet and deposition of the chemical in animal tissues.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan). 11(6): 445-448; 1970.* (6 references) (Japanese)

The distribution of lindane in tissues was investigated in rats fed diets containing 1, 10, or 100 ppm lindane. After 21 days livers of rats fed 1 ppm lindane contained 6 ppm lindane, kidney, 5 ppm, brain 4 ppm, and adipose tissue 3 ppm. Livers of rats fed 10 ppm for 21 days contained 30 ppm lindane, kidneys 15 ppm, brain 10 ppm, and adipose tissue 20 ppm. After 21 days on the ration containing 100 ppm lindane, rat livers contained 40 ppm lindane, kidney 120 ppm, brain 60 ppm, and adipose tissue 14 ppm. The lindane concentrations in various tissues appeared to reach an equilibrium level in 3-7 days in the rats fed at 100 ppm. The rise in tissue concentration was more gradual at the lower dosage levels; a slight drop was noted after the peak was attained in 2-3 wk. A marked depression of growth was produced by administration of a diet containing lindane at 100 ppm for 56 days, but growth in the other groups was not affected.

73-0695. Takeda, M.; Isobe, K.; Nigo, T.; Tanabe, H.; Kawashiro, I. (Nat. Inst. of Hyg. Sci., Kamiyoga, Setagaya, Tokyo, Japan). **Metabolic fate of organomercuric compounds (I). Reaction of organomercuric compounds with plant tissues.** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 12(3): 152-155; 1971.* (8 references)

Because of the extreme differences in toxicities between organic and inorganic mercury compounds, the behavior of phenylmercuric acetate (PMA) and methylmercuric chloride (MMC) was investigated in young plants. PMA lost its bactericidal activity immediately after addition to juices from cucumber fruits or broad bean plant leaves or in 1 and 2.5% ovalbumin. After standing for 24 hr in the juices or in ovalbumin solution, phenylmercuric derivatives, but not *o*- or *p*-oxyphenylmercuric, mercuric, mercurous, or methylmercuric derivatives were present. Mercury-free soybean sprouts grown in 100 ppm PMA solution contained inorganic as well as phenylmercuric derivatives. Inorganic mercury was also detected in wheat plants grown in PMA or MMC. Only phenylmercuric compounds were detected in rice bran.

73-0696. Isobe, K.; Takeda, M.; Tanabe, H.; Kawashiro, I. (Nat. Inst. of Hyg. Sci., Kamiyoga, Setagaya, Tokyo, Japan). **Metabolic fate of organomercuric compounds (II). Effect of thiol compounds on decomposition of organomercuric compounds.** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc.) 12(3): 156-159; 1971.* (4 references)

The effect of thiols and certain other compounds on the chemical decomposition of phenylmercuric acetate (PMA) was investigated. The recovery of PMA was reduced by exposure to dihydrothioctic acid, glutathione, and cysteine, in decreasing order of activity, under acidic conditions. The formation of inorganic mercury compounds paralleled the decrease in phenylmercuric acid recovered from solutions containing thiol compounds. Very little inorganic mercury was detected after exposure of PMA to solutions of carboxylic acids, mineral acid salts, and reducing sugars. Under slightly acid conditions glutathione accelerated the decomposition of alkylmercuric compounds about the same as it did PMA. Recoveries of mercurials were greatly affected by the molar ratio to glutathione in the case of *n*-propylmercuric chloride but not PMA. Degradation of mercurials in plants may be partially affected by the presence of thio groups and slightly acid conditions, but the reaction velocities observed in this study were too low to explain completely the decomposition of organomercuric derivatives in plant tissues.

73-0697. Takeda, M.; Isobe, K. (Nat. Inst. of Hyg. Sci., Kamiyoga, Setagaya, Tokyo, Japan). **Metabolic fate of organomercuric compounds (III). Studies on the decomposition of organomercuric compounds in wheat roots.** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 12(3): 160-163; 1971.* (2 references)

Studies on the in vitro degradation of organomercuric compounds in wheat roots revealed a lag phase followed by rapid decomposition of phenylmercuric acetate (PMA) and the four alkylmercurials tested. Each alkylmercurial had a different reaction rate. No lag time

was observed when wheat roots were pretreated with the compound to be tested, but decomposition was reduced considerably. Evidently the decomposition of mercurials was not due to substances secreted from the roots.

73-0698. Oshiba, K.; Kawakita, H. (Osaka Municipal Pub. Health Lab., Kita, Osaka, Japan). **[Interaction between toxicant and nutrition. (V) The fate of β-and γ-BHC in vivo following dietary protein levels.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Japan) 13(5): 383-387; 1972.* (16 references) (Japanese)

The effects of dietary protein levels on BHC deposited in organs and tissues were determined in 12 groups of rats. Four groups of six female Sprague-Dawley rats were fed a standard diet containing 10 ppm β-BHC for 28 days; another four were fed the diet containing 100 ppm γ-BHC for 14 days; others were fed just the standard diet for 28 days. After the feeding and when it was assumed that both BHC isomers had accumulated in the rat body, the diet was changed to include different amounts of protein, 5, 10, 20, and 30% casein for six days. Autopsy revealed that the deposited β-BHC in the liver, 47.8 μg/g tissue lipid, decreased to 41.2, 34.3, 29.6, and 25.3, respectively. The effect was not as marked in the other organs and tissues. Gamma-BHC also clearly decreased. The efficiency of decreasing the deposited BHC depended on the organs and tissues involved and the amount of protein intake. In a second experiment to determine whether dietary protein levels could be related to deposition, 12 groups of rats were fed diets containing different amounts of protein, 5, 10, 20, and 30% casein for 28 days. The diet for one third of the groups contained no BHC; another third received 10 ppm of γ-BHC (lindane); the last third received 10 ppm β-BHC. The deposition of γ and β-BHC in liver was markedly smaller as the protein in the diet increased: 54.8 μg/g with a 5% casein diet and 17 μg/g with 30% casein diet with γ-BHC; 94.3 μg/g with 5% and 34 μg/g with 30% casein diet with β-BHC. The same tendency was noticed in other organs and tissues. The above findings seem to be due to the induced activation of enzymes for dehydrochlorination of BHC in liver by the increased protein level; the acceleration of conjugation of glutathione and BHC metabolites; and the increased supply of SH groups of protein concerning the detoxication of foreign chemicals, suggesting a method of artificial detoxification of deposited BHC in the human body.

73-0699. Peardon, D. L.; Kilbourn, E. E.; Ware, Jr.; J. E. (Rohm and Haas Co., Philadelphia, PA). **New selective rodenticides.** *Soap Cosmet. Chem. Spec. 48(12): 70, 72, 74, 80, 82, 84; 1972.* (6 references)

A series of carbamates have been found to be selective rodenticides effective against mice, rats (including warfarin-resistant rats), and other rodents, but relatively innocuous to pets and birds. Bait acceptance is good and rodents die within 8 hr after a single feeding. The activity was discovered during a routine antiparasitic screening program. A > 200-fold difference was obtained between LD50 values for rats and chickens with one of the compounds. The same compound was fifty times more toxic to rats than to dogs. Rats consumed poisoned bait containing 10 times the effective dose. Good results have been obtained in all of the efficacy tests required by the Environmental Protection Agency.

73-0700. Zlateva, M.; Dishovska, Z. (Unified Sci. Res. Inst. Hyg. and Occup. Safety, Sofia, Bulgaria). **Klinikomorfologichni prouchvaniya vurkhu promenite v ustnata kukhinana beli plukhove sled khronichno zatravyane s lindan. [Clinical and morphological studies of the alterations of the oral cavity occurring in albino rats after chronic poisoning with lindane.]** *Stomatologiya (Sofia) 54(1): 8-13; 1972.* (6 references) (Bulgarian)

Intragastric administration of lindane to albino rats under chronic conditions produced a wide range of alterations in the parodontal tissue. Treatment with 1/20 LD50 (8.5 mg/kg) of lindane daily for two-months caused no histologically detectable damage. Rats receiving 1/100 LD50 (1.7 mg/kg) of lindane for six months had severe deterioration of the parodontal system including pathological gingival and osseous pockets as well as loosening and loss of teeth. Dystrophic changes of the periodontal membrane and of the alveolar bone were observed along with severe inflammatory processes in some of the papillae with osteoclastic resorption in the dental apex. The extent of pathological changes was closely related to the duration of exposure to lindane.

73-0701. Epstein, S. S.; Arnold, E.; Andrea, J.; Bass, W.; Bishop, Y. (Dept. of Pharmacol., Case Western Reserve Univ. Med. Sch., Cleveland, OH 44160). **Detection of chemical mutagens by the dominant lethal assay in the mouse.** *Toxicol. Appl. Pharmacol. 23(2): 288-325; 1972.* (20 references)

A total of 174 test agents, including pharmaceuticals, food additives, pesticides, and organic extracts of air and water pollutants was tested for mutagenicity in mice using the modified dominant lethal assay. In concurrent control populations the mean weekly pregnancy rate was 66% and exceeded 30% in 99% of all weeks; the distribution of weekly mean total implants per pregnancy was symmetrical around a peak of 11.5-11.9 and was never less than 8; mean early fetal deaths per pregnancy were <0.85 in 99.6% of weeks, and their distribution was highly asymmetrical. These parameters afforded the basis for screening the large body of test data. All agents with mean weekly values beyond control limits were subject to analysis of variance for test and concurrent control data in all replicate experiments. Less than 10% of all agents tested

were unequivocally mutagenic as determined directly by increased early fetal deaths per pregnancy and, in some instances, also indirectly by reduction in total implants per pregnancy; the majority of these are known alkylating agents. Additionally, about 5% of all agents tested yielded data which fell beyond control limits and which were significant at <5% by analysis of variance, but which, however, require further replication because of internal inconsistencies. (Author abstract by permission)

73-0702. Yasuda, M.; Maeda, H. (Dept. of Anatomy, Kyoto Prefect. Univ. of Med., Kamikyo, Kyoto, Japan). **Teratogenic effects of 4-chloro-2-methylphenoxyacetic acid ethylester (MCPEE) in rats.** *Toxicol. Appl. Pharmacol. 23(2): 326-333; 1972.* (15 references)

One of the phenoxyalkanoic acid herbicides, 4-chloro-2-methylphenoxyacetic acid ethylester (MCPEE), was mixed with food and given to pregnant Wistar rats from day 8 to 15 of gestation at levels of 40, 500, 1000 and 2000 ppm (about 2.7, 30, 60 and 100 mg/kg/day, respectively). Treatment with 2000 ppm of MCPEE resulted in decreased maternal weight gain and suppressed food consumption. No adverse fetal effect was noted at the 40 and 500 ppm levels. At the 1000 and 2000 ppm levels the fetal weight decreased, and the malformation rate increased as the dose increased. At the 2000 ppm level an increased resorption rate was observed. Major malformations produced were cleft palate, ventricular septal defect, and kidney anomalies. These results indicate that MCPEE at a high dose level is embryopathic in the rat. (Author abstract by permission)

73-0703. Blend, M. J.; Visek, W. J. (Dept. of Biol., Univ. of Detroit, Detroit, MI 48221). **Effects of low concentrations of dieldrin and chlormadinone acetate on canine prostatic fluid.** *Toxicol. Appl. Pharmacol. 23(2): 344-348; 1972.* (13 references)

Prostatic fluid was collected from 24 vasectomized mongrel dogs with cystopreputiostomies for 5 mo in each of two experiments; during this time dogs were fed 50 μg/kg body weight/day of chlormadinone acetate (CAP) and/or 15 μg/kg body weight/day of dieldrin. Collections were made for 1 hr under 0.7 mg/kg pilocarpine hydrochloride stimulation. Prostatic fluid volume and acid phosphatase activity decreased in CAP and CAP + dieldrin fed dogs. In the first experiment a significant hormone-pesticide interaction ($P < 0.05$) was observed at 4 mo when prostatic fluid volume per collection decreased 99% from pretreatment volumes with CAP and only 50% with CAP + dieldrin. Similar trends were observed in the second experiment although the hormone-pesticide interaction was not statistically significant ($P < 0.05$). An 80% decrease in prostatic secretion volume in dogs fed CAP was observed whereas only a 50% reduction occurred when CAP + dieldrin were fed. Again, acid phosphatase activity was decreased in CAP and CAP + dieldrin fed dogs but was unaltered in dogs fed dieldrin alone. Total solids content, pH, specific gravity, and osmolarity showed no significant differences corresponding to treatment in either experiment. (Author abstract by permission)

73-0704. Perlovskaya, Z. D.; Dubyagin, V. A.; Luzhnichenko, N. N. (Kirgiz. Sci. Res. Inst. Epidem., Microbiol. Hyg., Frunze, USSR). **K izucheniyu mekhanizma deystviya fenurona. [On the mechanism of action of fenuron.]** *Sov. Zdravookhr. Kirg. 6: 47-50; 1971.* (Russian)

The effect of daily 320 mg/kg-doses of fenuron on guinea pigs was studied in a 4-month experiment. Fenuron caused a 12.5% decrease in the erythrocyte count and a 13.2% decrease in the hemoglobin level as well as dysfunction of the endocrine system and blockade of SH groups. Both the diuresis and the 17-ketosteroid excretion decreased initially, after which diuresis normalized, and the 17-ketosteroid excretion jumped to 166.2% after 3.5 months. Increased sensitivity to experimentally produced hypoxia was observed. All the changes observed were relatively slight with tendency to normalization, which indicates the low toxicity of fenuron in subacute experiments.

73-0705. Johansson, D. (Author address not given). **Metoxyklor—ett av alternativen till DDT. [Methoxychlor—one of the possible substitutes for DDT.]** *Vaextskyddsnotiser 35(5-6): 63-65; 1971.* (12 references) (Swedish)

Animal and human toxicity tests of methoxychlor, a possible substitute for DDT, are reviewed. Considerably reduced acute and chronic toxicity, high rate of elimination, and little accumulation are the basic advantages of methoxychlor over DDT. The acute toxicity of methoxychlor is about 20-25 times lower than that of DDT with an oral LD50 value of 5,000-7,000 mg/kg. Even high doses fail to produce nervous symptoms such as paresthesia. The LD50 value for bees is about 24 μg. Elimination of 98% in 24 hr and of the total dose of methoxychlor in 11 days was observed in animal tests. The minimum intake to produce increased mortality in animals was 10,000 ppm. No organ damages and tumors due to methoxychlor were detected. A dose of 2 mg/kg, administered over two months, proved ineffective in a human toxicological test.

73-0706. Vulchovski, Ya.; Petrov, D.; Stoimenov, V, (Veterinary Med. Inst. of Infectious and Parasitic

Diseases, Sofia, Bulgaria). **Ostatuchni kolichestva Sevin v yaytsata, organite i tukanite na ptitsite. [Residual amounts of Sevin in the eggs, organs, and tissues of birds.]** *Vet. Med. Nauki 9(3): 83-90; 1972.* (7 references) (Bulgarian)

Several groups of one-year-old white chickens were treated p.o. with a single dose of carbaryl (Sevin) (250 or 500 mg/kg) or dusted with 6 g of a 5% carbaryl-containing powder to study its effects on blood cholinesterase levels and its retention in various tissues. Maximum cholinesterase inhibition (by 25% in the 250 mg/kg treated and by 32% in the 500 mg/kg treated group) occurred 3 hours after administration of the compound. Dusting produced no blood cholinesterase level changes. Normal enzyme levels were restored 36 hours after treatment. Maximum carbaryl retention occurred in the crop and muscular and glandular stomach tissues after 24 hours in both groups, and traces of carbaryl and 1-naphthol were ascertained 48 and 96 hours after 250 and 500 mg/kg administration, respectively. Total elimination of the compound occurred after 96 and 120 hours, respectively. No traces of carbaryl or its metabolites were found in heart, brain, skin or fatty tissues, indicating its fast breakdown and elimination rate and emphasizing the non-lipotropic character of the compound. Carbaryl residues in the egg yolk could be found up to four days after a single 500 mg/kg dose. Eggs treated with a 0.5% suspension of carbaryl contained an average residue of 0.115 mg/kg egg white while those treated with a 2% suspension reached 0.158 mg/kg levels of carbaryl in their white. Thirty days of storage at 4°C produced no alterations in the egg-white carbaryl content and no egg yolk contamination occurred. No carbaryl residues were found in eggs exposed to dusting with a 5% carbaryl containing powder.

73-0707. Lees, P. (Dept. of Physiol., The Royal Vet. College, Field Station, Hawkshead Ln., Brookmans Park, Harfield, Herts, England). **Pharmacology and toxicology of alpha chloralose: a review.** *Vet. Rec. 91(14): 330-333; 1972.* (11 references)

Pharmacological actions of α-chloralose include: both stimulation and depression of the central nervous system; serious impairment of respiratory function at doses sufficient to produce deep anesthesia; reduction of body temperature directly related to depth of anesthesia and probably body surface area to volume ratio. The rodenticide is metabolized in the body to chloral and then trichloroethanol, a CNS depressant which combines with glucuronic acid to be excreted in the urine. Birds and hedgehogs are more susceptible. However, hungry cats refused to eat α-chloralose bait formulations while dogs consumed 4 g/kg. Poisoning in cats has generally resulted from consumption of poisoned mice or birds. In acute overdosage the lethal action of the compound is generally due to CNS depression. Procedures for treating poisoning are primarily supportive and include restraint, maintenance of normal body temperature, evacuation of the stomach, and, if necessary, artificial ventilation of the lungs. Administration of drugs to counteract CNS depression or stimulation are not recommended.

73-0708. Johannes, H.; Kemmerling, W.* (A-2500 Baden bei Wien, Austria). **Chemische Krautbekaempfung. [Chemical aquatic weed control.]** *Wasser Boden 24(8): 235-237; 1972.* (16 references) (German)

Use, side effects, and toxicity of herbicides suitable for water weed control are reviewed. Benzonitriles, quaternary ammonium compounds such as paraquat, phenoxyacetic acids, halogenated aliphatic acids such as TCA and dalapon, urea derived compounds such as diuron, as well as triazine derivatives such as simazine and terbutryne are the herbicides suitable for the control of aquatic weeds. Except for quaternary ammonium compounds, these herbicides have low acute oral toxicity in warm-blooded organisms and are not hazardous to fish. The metabolites of these herbicides are not more toxic than the original herbicides. Such herbicides in water may cause reduced oxygen content due to the destruction of algae as well as certain shifts in the plankton composition.

73-0709. Schoebel, A.; Tscheu, M.; Wotzka, J. (Author address not given). **Toxikologische Pruefung und biochemisches Abbauverhalten von Abwaessern aus der Herbizidproduktion. [Toxicological analysis and biochemical decomposition properties of waste waters from herbicide production.]** *Wasserwirt.-Wassertech. 22(10): 342-346; 1972.* (18 references) (German)

The toxicity of waste waters from 2,4-D, dalapon, MCPA, and MCPB in *Ankistrodesmus falcatus (Corda) Ralfs* and in *Cyprinus carpio* and the biochemical decomposition properties of such waste waters were studied. The dalapon, 2,4-D, MCPA, and MCPB waste waters contained 2,2-dichloropropionic acid, 2,4-dichlorophenoxyacetic acid, 2-methyl-4- chlorophenoxyacetic acid, and 2-methyl-4-chlorophenoxybutyric acid, respectively, as effective agents. The waste water from 2,4-D production had the highest toxicity and that from MCPB production the lowest in *Ankistrodesmus falcatus* with dalapon and MCPA waste waters having intermediate toxicity values. Dalapon and MCPA waste waters diluted at the respective ratios of 1:20 and 1:10-50 were lethal for *Cyprinus carpio* in a 96 hr test. The corresponding threshold concentrations of the above waste waters were 1:50 and 1:250. Biological decomposition tests adopting the activated sludge method revealed the possibility of treating dalapon, 2,4-D, and MCPB waste waters by biological waste water treatment processes.

73-0710. Ising, E. (Erprobungsstelle 52 d. Bw., Munster, Germany). **Rueckstaende des Thiophosphorsaeureesters Bromophos (Cela) im Kot von Meerschweinchen nach**

oraler Applikation. [Residues of bromophos (Cela) in guinea pig feces after oral administration.] *Z. Angew. Zool. 58(4): 465-473; 1971.* (20 references) (German)

Insect test cultures failed to grow in laboratories previously used for housing guinea pigs. Since the guinea pigs were used in chronic toxicity tests with bromophos, the possibility of residual bromophos in the room was considered. When guinea pigs were fed carrots spiked with bromophos, insecticidally active feces were excreted between the second and the sixth hours after feeding. A high proportion of administered bromophos was excreted with the feces. Insecticidal activity was determined by mixing fresh guinea pig feces with the food supplied to mosquito larvae incubated in Petri dishes. Dried feces from bromophos feeding experiments, stored for 2½ years, retained a high level of insecticidal activity. Experiments with insect parasites can be falsified either by direct contact between insects and feces (i.e., if the pelts of experimental animals are contaminated with feces) or by dispersion of bromophos-containing particles into the air during the handling of animals, cages, or bedding.

73-0711. Schulz, D. (Inst. fuer Vet. Pathol., Freie Univ. Berlin, Berlin, Germany). **Proliferative Endokarditis am Herzen von Karpfen nach Einwirkung des Herbizids "Karmex." [Proliferative endocarditis in the heart of the carp after exposure to the herbicide Karmex.]** *Zentralbl. Veterinaermed. 19(5): 390-406; 1972.* (59 references) (German)

The effects of prolonged exposure to Karmex (80% diuron powder) were studied in one-summer-old carp maintained in tanks of water containing diuron at 12, 6, 0.5, 0.2, and 0.1 ppm. Fish maintained in 1 m-deep experimental pond treated with 1 kg/ha of Karmex (ca. 2 ppm in water) were also examined. Fish mortality in the highest-dosage group was first observed on the 16th day of the experiment and on the 23rd day in the second group. All fish in these two groups died or were killed in a moribund state. Fish in the lower-dosage groups displayed loss of appetite, unresponsiveness, and weight loss, but survived until sacrificed for examination after 40 days. Fish in the two highest-dosage groups displayed severe congestive degeneration of kidneys, liver, and gall bladder, and proliferative changes in the heart. In the lower-dosage groups and in the pond fish proliferative degeneration of endocardium, pericardium, and myocardium was the most pronounced finding. The pond fish in particular developed extensive granulomas on the heart valves. Degenerative changes in the gonads were also most severe in the higher-dosage groups.

73-0712. Bromberg, A. I.; Potsheba, T. L.; Pozin, Z. S.; Brikman, L. I.; Volkova, A. P.; Voronkina, T. M.; Kamenskiy, I. V. (Moscow City Disinfection Station, Moscow, USSR). **Novaya forma primeneniya khlorofosa–preparat "Insektopolimer." [New application form of trichlorfon–the preparation "Insektopolimer."]** *Zh. Mikrobiol. Epidemiol. Immunobiol. 49(7): 133-138; 1972.* (11 references) (Russian)

Toxicity tests of a new "Insektopolimer" preparation (5-10 parts of trichlorfon dissolved in water containing 0.5-2 parts of polyvinyl alcohol) are described. After the water evaporates from the preparation applied to surfaces to be treated, an inconspicuous, durable coating containing crystallized trichlorfon is left behind. The polyvinyl alcohol admixed to trichlorfon minimizes the insecticide losses and considerably increases the stability of trichlorfon for long-term effect. The permanent inhalation of trichlorfon vapors by warm-blooded animals over 2-3 weeks in a chamber treated with 12 g of trichlorfon/m^2 caused no poisoning symptoms. The trichlorfon concentration in indoor air fell to below the maximum allowable concentration of 0.5 mg/m^3 shortly after application. The "Insektopolimer" with 5 or 10% of trichlorfon should be used at a maximum expenditure of 10 g/m^2.

See also 73-0502 73-0507 73-0547 73-0562 73-0590 73-0599 73-0600 73-0621 73-0626 73-0631 73-0631 73-0633

73-0713. Murano, A. (Takarazuka Res. Lab., Sumitomo Chem. Ind. Co., Ltd., Takarazuka, Japan). **Determination of the optical isomers of insecticidal pyrethroids by gas-liquid chromatography.** *Agr. Biol. Chem. 36(12): 2203-2211; 1972.* (25 references)

Determination of the optical isomers of insecticidal pyrethroids was carried out by gas-liquid chromatography. Chrysanthemic acid, which was obtained during hydrolysis of the corresponding pyrethroids, was esterified with *d*- or *l*-2-octanol and the diastereoisomer ester derivatives were resolved on a column of Chromosorb W coated with 10% QF-1. All four isomers, *d-trans, l-trans, d-cis* and *l-cis* chrysanthemic acids, could be separated. These compounds were not isomerized during hydrolysis, esterification, or gas liquid chromatographic operations and their proportions were accurately determined from their peak area ratios. (Author abstract by permission)

73-0714. Abramson, F. P. (Dept. of Pharmacol. and Pathol., George Washington Univ., Sch. of Med., Washington, DC 20005). **Applications of mass spectrometry to trace determinations of environmental toxic materials.** *Anal. Chem. 44(14): 28A-35A; 1972.* (24 references)

Advantages which mass spectrometry offers over traditional analytical methods for environmental contaminants include: relatively uniform high sensitivity for all volatilizable materials; excellent selectivity by use of specific ion detection or high resolution; easy identification of unexpected materials; and freedom from false positive results. Reference spectra at the 100 ng/μl level for lindane, heptachlor, aldrin, chlordane, dieldrin, *p,p'*-DDE, *o,p'*-DDD (TDE), endrin, *o,p'*-DDT, *p,p'*-DDD, and *p,p'*-DDT show that the chemical integrity important to mass spectrometric analysis of these chlorinated pesticides is preserved. The separating power of the MS was demonstrated with a mixture of methyl mercuric chloride, Arochlor 1260, and lindane. Data processing in conjunction with MS is useful for organochlorines and particularly for polychlorinated biphenyls. Although the initial investment in MS is considerable, the diversity of analyses, ease of preparation, and higher information content provide overall economy.

73-0715. Hebel, R. (Staatl. Veterinaeruntersuchungsamt, Krefeld, Germany). **Zur Systematik von Rueckstandsanalysen in Lebensmitteln. [Systematic approach to analysis of residues in foods.]** *Arch. Lebensmittelhyg. 23(8): 165-169; 1972.* (8 references) (German)

Trace analysis is a distinct discipline within analytical chemistry dealing with the detection of substances present in at least a 10,000-fold excess of a matrix material. Chemical residues in foods are present in the lower ppm and ppb range. Residue analyses comprise four steps: sample collection, sample preparation and concentration of trace materials, a detection step, and evaluation and interpretation of results. The preferred methods of detection in the trace range are physical methods since these generally permit characterization and quantitation in a single step. The toxicologist, particularly the veterinary toxicologist, is generally responsible for the interpretation of analytical results. Important classes of residues in food at present include drugs, pesticides, animal growth accelerators, and materials (such as heavy metals) from the natural environment. Category boundaries may be crossed in some cases; for example, technical DDT formulations contain about 20% *o,p'*-DDT, which has estrogenic as well as insecticidal activity.

73-0716. Liebmann, R.; Hempel, D.; Heinisch, E. (Forschungszent. fuer Pflanzenschutzm., VEB Fahlberg-List, Magdeburg, DDR). **Beitraege zur Analytik von Toxaphen. [Survey of toxaphene analysis.]** *Arch. Pflanzenschutz 7(2): 131-150; 1971.* (31 references) (German)

Toxaphene has been known for 20 years and is expected to become increasingly important with curtailment of DDT use, particularly because of its limited accumulation in mammals. Nevertheless, analytical methods for toxaphene are presently inadequate. Analysis is complicated by the fact that toxaphene consists of an undefined mixture of chlorinated terpenes. Various elemental chlorine determination methods (Stepanow, Schoninger, and sodium biphenyl methods) are used for analysis of toxaphene formulations. Colorimetric procedures for the determination of toxaphene residues in foods include the method of Graupner and Dunn (using diphenylamine and zinc chloride) and the ethylcyanoacetate method. In colorimetric procedures careful extraction of the active ingredient is required to remove interfering food pigments. Column chromatography and thin-layer chromatography are often used as separation methods, and thin-layer chromatography can be used for rapid toxaphene detection as well. Complete specifications for all the procedures mentioned, as well as for determination of toxaphene in combination preparations, are given.

73-0717. Spengler, D.; Jumar, A. (Forschungszentr. Pflanzenschutz-und Schaedlingsbekaepmfungsm., VEB Fahlberg-List, Magdeburg, DDR). **Bestimmung der Rueckstaende von Carbamat- und Harnstoffherbiziden im Boden. [Determination of carbamate and urea herbicide residues in soil.]** *Arch. Pflanzenschutz 7(2): 151-159; 1971.* (12 references) (German)

Carbamate and urea herbicide residues in soil can be determined by a colorimetric method or a semiquantitative thin-layer chromatographic method. Because of the presence of large numbers of interfering components in the soil specimens, the acetone soil extracts of the herbicides must be hydrolyzed and the resulting anilines cleaned up by steam distillation prior to coupling with

thymol, forming azo dyes. The dyes are passed over a cellulose column and eluted with aqueous ammonia, then the optical density of the appropriate dye band determined. In the thin-layer chromatographic estimation of the herbicides, components from unpurified soil extracts prevent the formation of clean spots of active ingredients. Acidified acetonic soil extracts can advantageously be cleaned up by precipitation with sodium tungstate. The herbicides are reextracted from the filtrate with chloroform and the concentrated extract applied to the thin-layer plate. Nearly 100% recovery after cleanup is attained with herbicides which are fairly water-soluble. Results of the colorimetric and TLC methods after application of proximpham to test fields at 2, 4, or 6 kg/ha were in good agreement, and the TLC method has the additional advantage of usefulness in case of combination preparations. About half of the proximpham initially present remained after one week and one-tenth remained after three weeks.

73-0718. Thier, H.-P. (Inst. fuer Angewandte Chemie, Univ. Erlangen-Nuernberg, Erlangen, Germany). **Analysengang zur Ermittlung von Pestizid-Rueckstaenden in Pflanzenmaterial. 1. Teil. [Analytic process for the determination of pesticide residues in plant material. Part 1.]** *Deut. Lebensm.-Rundsch. 68(11): 345-350; 1972.* (German)

Procedures for the extraction, separation, and chromatographic determination of pesticide residues in plant materials are described. The extraction from fruits, vegetables, and cereals is done best with acetonitrile. The acetonitrile extract diluted with water is shaken with a mixture of eight parts of petroleum ether and two parts of methylene chloride, after which the organic phases obtained are combined, diluted with water, and distilled. In another method the filtrate from the acetonitrile extract is treated with a similar mixture of petroleum ether and methylene chloride to which a small amount of 10%-sodium chloride solution is added. The lower aqueous phase is then shaken with the same mixture of petroleum ether and methylene chloride, after which the homogeneous solution obtained through combining all organic phases is diluted with water and distilled. The extract obtained by any of the two procedures is purified by means of column chromatography on Florisil from which elution is done by different solutions. Two chromatographic columns with silicone derivatives of opposite polarity or one column containing a mixture of two different stationary phases are used for the separation. The conditions for the gas-chromatographic analysis are carrier gas (argon + methane) flow rate of 120 ml/min, injection temperature of 220 C, column temperature of 200 C, detector temperature of 300 C, and about 2 min retention time for dieldrin.

73-0719. Takeshita, R.; Akagi, H.; Sakagami, Y. (Inst. of Pub. Health, Tokyo, Japan). **[Thin-layer chromatography of chlorinated phenoxy herbicides on polyamide layers.]** *Eisei Kagaku (J. Hyg. Chem.) 17(5): 337-340; 1971.* (13 references) (Japanese)

The separation and identification of chlorinated phenoxy herbicides by chromatography on polyamide thin layers was examined. Out of the fourteen solvent mixtures tested, good separation was attained with mixtures of nonpolar solvents and acetic acid such as benzene:acetic acid 9:1 and dichloromethane:acetic acid 19.5:0.5. Chromatoplates were visualized by spraying with *o*-toluidine, *o*-dianisidine, or silver nitrate, and examined under UV light. Various herbicides were detectable at 1-4 μg.

73-0720. Baluba, S. A. (Tselinograd District Bureau of Forensic Medicine, Tselinograd, USSR). **Opredeleniye khlorofosa pri issledovanii biologicheskogo materiala. [Determination of trichlorfon in biological materials.]** *Farmatsiya (Moscow) 20: 82-83; 1971.* (Russian)

A method for the determination of trichlorfon residues in putrid biological materials, particularly in liver, is described. The extract obtained in subsequent stages by means of acetic acid, chloroform, and acetone is dissolved in ethanol for horizontal paper electrophoresis using a 30% solution of acetic acid as electrolyte. After electrophoresis for one hour, traces of trichlorfon appear as greenish-blue fluorescent spots in ultraviolet light. The electropherograms are then processed by different procedures using resorcinol with sodium carbonate, potassium hydroxide in methanol with silver nitrate in nitric acid, benzidine in acetone with alkaline solution of hydrogen peroxide, and oxidizing agents such as potassium permanganate, sulfuric acid, and nitric acid as reagents for further analysis. Quantitative determination is done by polarography. The output for samples containing 10 and 1 mg of trichlorfon is 50% and 10.8%, respectively.

73-0721. Seehofer, F.; Nesemann, E. (Forschung und Entwicklung, BAT Cigarettenfabriken GmbH, Hamburg, Germany). **Pesticidrueckstandsprobleme bei Nutzpflanzen. Mit eigenen Beitraegen aus der Tabakanalytik. [Pesticide residue problems in commercial plants. Including original contributions on tobacco analysis.]** *Fresenius Z. Anal. Chem. 261(4-5): 359-369; 1972.* (141 references) (German)

Following a review of the techniques and equipment used in the determination and interpretation of pesticide residue data, specific problems of pesticide residues in tobacco are discussed. Pesticides have been used in large quantities on tobacco since the product has a high inherent value in terms of labor and capital applied. The importance of residues in tobacco is reduced by tobacco processing, by the burning of the tobacco during smoking, and by the relatively small quantity of tobacco products consumed compared to foods. The backbone of the tobacco analyses used by the German

cigarette industry is the gas chromatograph, operated with standardized columns, simple temperature programs, and a variety of detectors. Standardization permits a high degree of reproducibility from one laboratory to another. Selected pesticide residue values from field trials and monitoring studies in Europe and elsewhere are presented, along with data on the reduction in pesticide content of tobacco during drying and the transfer of pesticides into the main stream smoke. In a shipment of tobacco from 1971, the DDT contents ranged from less than 1 to 30 ppm, and the dithiocarbamate contents, from 50 to 2000 ppm. Improved analytical methods and the resulting efforts led to a ten-fold reduction in pesticide residues in tobacco in recent years.

73-0722. Thielemann, H. (DDR-36 Halberstadt, Wasserturmstrasse 10, DDR). **Zur duennschicht-chromatographischen Trennung und Identifizierung herbizidwirksamer Substanzen an Fertigfolien. [Thin-layer chromatographic separation and identification of herbicidal substances on prepared (aluminum) foil.]** *Fresenius Z. Anal. Chem. 262(3): 192; 1972.* (2 references) (German)

The thin-layer chromatographic separation and identification of 2,4-dichlorophenoxypropionic acid, 1-methyl-3-chloro-4-phenoxybutyric acid, 1-methyl-3-chloro-4-phenoxypropionic acid, 1-methyl-3-chloro-4-phenoxyacetic acid, 2,4-dichlorophenoxyacetic acid, and 2,4-dichlorophenoxybutryic acid by means of ready-made foils is described. A mixture of 40 parts of benzene with 60 parts of acetone (80 parts of benzene with 20 parts of acetone for 1-methyl-3-chloro-5-phenoxybutyric acid and for 2,4-dichlorophenoxybutyric acid) is used as solvent. After the foils are dried in air they are sprayed with 0.01%-solution of Rhodamine B in ethanol. Stable violet spots against a rose background appear following ultraviolet irradiation for 5 min.

73-0723. Williams, I. H.; Brown, M. J.; Finlayson, D. G. (Canada Dept. of Agr., Vancouver 8, British Columbia, Canada). **Determination of residues of fensulfothion and its sulfone in muck soil.** *J. Agr. Food Chem. 20(6): 1219-1221; 1972.* (8 references)

A method is described for determining fensulfothion (*O,O*-diethyl *O*-[*p*-(methylsulfinyl)phenyl] phosphorothioate) and its sulfone in muck soils, using Soxhlet extraction with a 9:1 chloroform-ethanol mixture. Cleanup and fractionation was on an alumina-silica gel column and determination was by gas chromatography with flame-photometric detection. Optimum recoveries were obtained when the moisture content of the soil was between 20 and 60%. Recoveries from soils fortified at 0.1, 1.0, and 10.0 ppm were 80% or greater. Residues in soils field-treated with fensulfothion for protection against carrot rust fly ranged from 19.9 to 90.5 ppm for the parent compound and from 5.9 to 21.8 ppm for its sulfone. (Author abstract reprinted by permission of the American Chemical Society)

73-0724. Newsom, H. C.; Mitchell, E. M. (U.S. Borax Res. Corp., Anaheim, CA 92801). **Determination of dinitramine residues in soil and plant tissue.** *J. Agr. Food Chem. 20(6): 1222-1224; 1972.*

Dinitramine (N^3,N^3-diethyl-2,4-dinitro-6-trifluoromethyl-*m*-phenylenediamine; tested under the names diethamine and USB 3584; trade name Cobex) is an effective new selective herbicide used for control of a wide variety of weeds in soybeans and cotton. An analytical method for determining residual quantities of this material has been developed which involves methanol extraction, methylene chloride partitioning, Florisil column chromatography, and measurement by electron capture gas chromatography. The method is applicable to soil, forage, and crop samples, and it gives good recoveries down to 0.01 ppm without subtraction of blanks. An alternative method utilizes the same extraction and clean-up procedure, but with alkali flame ionization detection. The latter method also is quantitative at 0.01 ppm. Application of these procedures to samples from field test plots demonstrated that dinitramine is readily degraded by environmental conditions over the growing season. Soil residues fell below 10% of application levels in about 100 days, while mature plant tissue and crop samples contained less than 0.01 ppm of dinitramine. (Author abstract reprinted by permission of the American Chemical Society)

73-0725. White, E. R.; Kilgore, W. W. (Dept. of Environ. Toxicol., Univ. of California, Davis, CA 95616). **Determination of systemic MBC residues in food crops treated with benomyl fungicide.** *J. Agr. Food Chem. 20(6): 1230-1232; 1972.* (6 references)

A procedure for the determination of benomyl [methyl 1-(butylcarbamoyl)-2-benzimidazole carbamate] and its decomposition product, MBC (methyl 2-benzimidazole carbamate), on various food crops is described. The compounds are first extracted with benzene and partitioned into 0.1 *N* hydrochloric acid. The acidic layer is washed several times with chloroform and then neutralized to pH 7.8-8.2 with concentrated sodium hydroxide. The single residual product, MBC, is partitioned into ethyl acetate, concentrated by evaporation, and subsequently developed on a commercially prepared thin-layer chromatogram containing a fluorescent indicator. The spot on the developed chromatogram having the same retention time as the standard methyl 2-benzimidazole carbamate is extracted with methanol and measured in an ultraviolet spectrophotometer at 287 mμ. The lower limit of sensitivity for this method is 0.05 ppm. Overall average recovery of benomyl residues obtained from fortified control samples was 87%. Recovery from grown-in labeled MBC

residues averaged 83%. (Author abstract reprinted by permission of the American Chemical Society)

73-0726. Stanley, C. W.; Thornton, J. S.; Katague, D. B. (Chemagro Div. of Baychem Corp., Kansas City, MO 64120). **Gas chromatographic method for residues of Baygon and metabolites in plant tissues.** *J. Agr. Food Chem. 20(6): 1265-1269; 1972.* (6 references)

A gas chromatographic method for the determination of Baygon (*o*-isopropoxyphenyl *N*-methylcarbamate, propoxur) and its metabolites in plants has been developed. The residues are removed from the plant sample by successive blending with acetone and chloroform. Baygon and the water-soluble conjugates are separated from each other and cleaned up separately. The conjugated metabolites are released by enzyme hydrolysis before the cleanup. Detection is by electron capture gas chromatography of the trichloroacetyl derivatives. (Author abstract reprinted by permission of the American Chemical Society)

73-0727. Stanley, C. W.; Thornton, J. S.* (Chemagro, Div. of Baychem Corp., Kansas City, MO 64120). **Gas chromatographic method for residues of Baygon and its major metabolite in animal tissues and milk.** *J. Agr. Food Chem. 20(6): 1269-1273; 1972.* (9 references)

A gas chromatographic method for the determination of Baygon (*o*-isopropoxyphenyl *N*-methylcarbamate) (propoxur) and its major metabolite, *o*-hydroxyphenyl *N*-methylcarbamate, in animal tissues and milk has been developed. The residues are extracted from animal tissues and milk by successive blending with acetonitrile and hexane. Baygon and the conjugated metabolite are separated from each other and cleaned up individually. The metabolite is released from its conjugate by acid hydrolysis before cleanup. Detection is by electron capture gas chromatography of the trichloroacetyl derivatives. (Author abstract reprinted by permission of the American Chemical Society)

73-0728. Lombardo, P.; Pomerantz, I. H.; Egry, I. J. (Div. of Chem. Technol., Food and Drug Admin., Washington, DC 20204). **Identification of photoaldrin chlorohydrin as a photoalteration product of dieldrin.** *J. Agr. Food Chem. 20(6): 1278-1279; 1972.* (6 references)

A photoproduct produced by the thin film irradiation of dieldrin with germicidal lamps has been isolated and identified. Based on GLC, IR, NMR, MS, and melting point comparisons with independently synthesized material, the product is considered to be a chlorohydrin derived from photodieldrin. The isolation and synthesis of the chlorohydrin is described. (Author abstract reprinted by permission of the American Chemical Society)

73-0729. Schroeder, R. S.; Patel, N. R.; Hedrich, L. W.; Doyle, W. C.; Riden, J. R.; Phillips, L. V. (Gulf Res. and Development Co., Merriam, KS 66202). **Analytical method for determination of residue levels of hydroxycyprazine (2-hydroxy-4-cyclopropylamino-6-isopropylamino-*s*-triazine) in corn.** *J. Agr. Food Chem. 20(6): 1286-1288; 1972.* (9 references)

An analytical procedure for determination of residue levels of hydroxycyprazine (2-hydroxy-4-cyclopropyl-amino-6-isopropylamino-*s*-triazine) in corn silage and grain has been developed. The procedure is based on extraction and separation of hydroxycyprazine, followed by its conversion to cyprazine (a chlorotriazine), which subsequently is determined gas chromatographically. (Author abstract reprinted by permission of the American Chemical Society)

73-0730. Chau, A. S. Y.; Terry, K. (Inland Waters Branch, Dept. of the Environ., Ottawa, Ontario, Canada K1Y 2P5). **Confirmation of pesticide residue identity. IV. Derivative formation in solid matrix for the confirmation of α- and β-endosulfan by gas chromatography.** *J. Ass. Offic. Anal. Chem. 55(6): 1228-1231; 1972.* (8 references)

The new solid matrix derivation procedure has now been applied to the confirmation of endosulfan residue identity. This procedure involves the conversion of endosulfan to the same diacetate in alumina impregnated with sulfuric acid and acetic anhydride. Comparable yield of the diacetate was obtained from the present approach and from a previously reported acetylation method. The advantage of the present method over the reported procedure is also discussed. (Author abstract by permission)

73-0731. Chau, A. S. Y. (Canadian Cent. of Inland Waters, Dept. of the Environ., Water Quality Div., Burlington, Ontario, Canada). **Confirmation of pesticide residue identity V. Alternative procedure for derivative formation in solid matrix for the confirmation of α- and β-endosulfan by gas chromatography.** *J. Ass. Offic. Anal. Chem. 55(6): 1232-1238; 1972.* (32 references)

A quick and sensitive new method has been developed for the confirmation of α- and β-endosulfan by conversion to the same ether in a solid matrix. It seems by far to be the most sensitive method reported to date for positive identification of α- and β-endosulfan. As little as 0.005 ppm of the parent compounds in a 10 g fish or sediment extract and 0.003 ppb in a 2 l. water extract can be routinely confirmed.. (Author abstract by permission)

73-0732. Norman, S. M.; Fouse, D. C.; Craft, C. C. (Market Quality Res. Div., U.S. Dept. of Agr., Pomona, CA 91769). **Thin-layer chromatographic separation and spectrophotofluorometric determination of thiabendazole residues on and in citrus.** *J. Ass. Offic. Anal. Chem. 55(6): 1239-1244; 1972.* (7 references)

A thin-layer chromatographic (TLC)-spectrophotofluorometric method was developed for quantitative determination of thiabendazole on and in citrus. Whole citrus fruits are surface stripped with ethyl acetate and a concentrated aliquot is separated by TLC. Whole-fruit blends, juice, oil, and dried citrus pulp cattle feed are extracted with ethyl acetate, partitioned into 0.1*N* HCl, and then partitioned into ethyl acetate-chloroform from an aqueous basic solution prior to TLC. Thiabendazole is eluted from the chromatogram with acidified methanol and the intensity of fluorescence is compared to standards similarly chromatographed and measured. The method is accurate in determining thiabendazole content ranging from 0.2 to 6 ppm. Recoveries of thiabendazole from fortified samples were 95-99% for surface strippings and 70-91% for rind and pulp extractives. No significant difference was found between residue values determined by the TLC method and by the Merck method. TLC eliminates citrus constituents which occasionally interfere with the Merck method. (Author abstract by permission)

73-0733. McGill, A. E. J.; Robinson, J.*; Stein, M. (Shell Res. Ltd., Tunstall Lab., Sittingbourne, Kent, England). **Methods of estimating dietary exposure of the general population to organochlorine insecticide residues: diet analyses 1965-1967.** *J. Ass. Offic. Anal. Chem. 55(6): 1245-1258; 1972.* (55 references)

The amounts of organochlorine insecticides in the diet of the general population of Great Britain have been estimated by analyzing representative whole cooked meals. The methods of quantitation are briefly outlined and techniques suitable for qualitative confirmation of identity of these compounds are discussed. The estimated dietary intakes are compared with the food in other dietary surveys and, in the case of dieldrin, they are also compared with the amounts calculated from the concentrations of this compound in body fat and blood. The dietary intakes of dieldrin in 1965-1966 and 1967 were estimated to be 0.00030 and 0.00020 mg/kg/day, respectively, the difference being statistically significant. The maximum estimated intakes of total equivalent DDT were 0.0011 and 0.0014, respectively. (Author abstract by permission)

73-0734. Frei, R. W.; Lawrence, J. F. (Trace Analysis Res. Cent., Dept. of Chem., Dalhousie Univ., Halifax, Nova Scotia, Canada). **Determination of Matacil and Zectran by fluorigenic labeling, thin layer chromatography, and in situ fluorimetry.** *J. Ass. Offic. Anal. Chem. 55(6): 1259-1264; 1972.* (21 references)

The fluorigenic labeling of Matacil (4-dimethylamino-*m*-tolyl *N*-methylcarbamate) (aminocarb) and Zectran (4-dimethylamino-3,5-xylyl *N*-methylcarbamate) with dansyl chloride (1-dimethylamino-naphthalene-5-sulfonyl chloride) results in three fluorescent derivatives; labeling with NBD-Cl (4-chloro-7-nitrobenzo-2,1,3-oxadiazole) produces two fluorescent derivatives for each carbamate. All five can be separated by thin layer chromatography (TLC). These derivatives are identified by nuclear magnetic resonance, infrared, and fluorescence spectroscopy, aided by TLC data. The carbamates are hydrolyzed in dilute base and the resulting amine or phenol hydrolysis products react with the labeling reagents. The derivatives are analyzed by TLC and in situ fluorimetry with a spectrophotometer in the fluorescence mode and a spectrophotofluorometer with the thin layer scanning accessory. Reactions, fluorescence phenomena, and chromatographic properties of the derivatives are investigated for evaluation of the method as a quantitative technique. A reproducibility of 3-5% relative standard deviation can be expected in the concentration range from 15 to 300 ng/spot for derivatives of the two labeling procedures. The dansyl derivatives are instrumentally detectable as low as 1 ng/spot while the NBD derivatives may be detected at concentrations of less than 0.5 mg/spot. (Author abstract by permission)

73-0735. Maini, P.; Collina, A. (Pestic. Residues Lab., Centro Esperienze e Ricerche, Soc. Italo Americano Prodotti Antiparassitari, Bologna, Italy). **Residues of chlorpyrifos insecticide in various crops. I. Sweep co-distillation method for extraction and clean-up.** *J. Ass. Offic. Anal. Chem. 55(6): 1265-1269; 1972.* (21 references)

The sweep co-distillation technique was expanded for application to chlorpyrifos insecticide residues in various substrates. The published technique was followed without significant changes for samples of lettuce, carrot, sugar beet (leaves and roots), potato, apple, onion, cauliflower, orange (pulp and peel), corn grain, and agricultural soil. Recoveries ranged from 74.2 to 100.7% at the fortification level of 0.1 ppm. Recovery tests were carried out also at levels ranging from 0.05 to 1.0 ppm for some crop samples obtained from trial field tests, with good results. For tobacco samples, some changes in the extraction and cleanup procedure were introduced to avoid some GLC interferences, but recoveries were low. Low recoveries were also obtained from the unmodified method and from the extraction, partition, and Florisil cleanup method. (Author abstract by permission)

73-0736. MacNeil, J. D.; Frei, R. W.*; Safe, S.; Hutzinger, O. (Trace Analysis Res. Centre, Dept. of Chem., Dalhousie Univ., Halifax, Nova Scotia, Canada). **Electron donor-acceptor complexing reagents in the analysis of pesticides. V. The analysis of pesticide decomposition products via electron donor-acceptor complexes; photolysis of methoxychlor.** *J. Ass. Offic. Anal. Chem. 55(6): 1270-1275; 1972.* (18 references)

Experiments are reported on the photolytic decomposition of the pesticide methoxychlor upon ex-

posure to different types of artificial light and sunlight. A number of products have been isolated, including methoxychlor olefin, DME, dimethoxybenzophenone isomers, methoxybenzoic acid isomers, and others. The decomposition of methoxychlor and the appearance of methoxychlor olefin and DME have been followed quantitatively by determining these compounds as their π-complexes with the electron-acceptor reagent 2,4,7-trinitro-9-fluorenone in situ on silica gel thin layer chromatograms by reflectance spectroscopy. Compounds have been identified by TLC followed by mass spectrometry and other techniques. The advantages of the nondestructive electron-acceptor sprays for these and similar analytical applications are demonstrated. (Author abstract by permission)

73-0737. El-Dib, M. A.; Aly, O. A. (Water Pollut. Contr., Nat. Res. Centre, Dokki, Cairo, Egypt). **Colorimetric determination of phenylamide pesticides in natural waters.** *J. Ass. Offic. Anal. Chem. 55(6): 1276-1279; 1972.* (23 references)

Phenylamide pesticides include a great variety of compounds such as *N*-phenylcarbamates, phenylureas, and anilides. A simple, rapid, and sensitive colorimetric method for their determination in natural waters has been proposed. Phenylamides are rapidly hydrolyzed in acid medium at elevated temperature (150-155°C), yielding the corresponding anilines. The latter compounds are diazotized and coupled with 1-naphthol to yield intensely colored azo dyes. Both phenylamides and aniline derivatives can be determined in a sample in the presence of each other. The method is sensitive to 0.02 mg/l.; coefficients of variation range between 6.0 and 0.2, according to the amount of phenylamide pesticide being measured. (Author abstract by permission)

73-0738. Steller, W. A.; Pasarella, N. R. (Agr. Div., American Cyanamid Co., Princeton, NJ 08540). **Gas-liquid chromatographic method for the determination of dimethoate and dimethoxon residues in plant and animal tissues, milk, and eggs.** *J. Ass. Offic. Anal. Chem. 55(6): 1280-1287; 1972.* (16 references)

Residues of both dimethoate and dimethoxon are extracted from finely chopped plant or animal tissues with methylene chloride. The resulting extract is concentrated and cleaned up by silica gel chromatography, if necessary. The concentrate is injected into a gas chromatograph equipped with either a flame photometric or an alkali flame ionization detector and a column containing 11% DC-200 on 60-80 mesh Gas-Chrom Q/0.01% Versamid 900. Recovery values for dimethoate and dimethoxon added at 0.002-1.0 ppm ranged from 56 to 112% with an overall average of 89.2% for dimethoate and from 60 to 127% with an overall average of 81.9% for dimethoxon. The lower limit at which recovery values were obtained for both compounds was 0.05 ppm for plant tissues, 0.02 for animal tissues and eggs, and 0.002 for milk. Residue values obtained for animal tissues involving both exposure and feeding studies and for field-treated plant samples are also presented. (Author abstract by permission)

73-0739. Heath, R. R. (Insect Attractants Lab., U.S. Dept. of Agr., P.O. Box 14565, Gainesville, FL 32601). **Rapid method for analyzing resmethrin content in formulations and determining the *cis-trans* ratios.** *J. Ass. Offic. Anal. Chem. 55(6): 1328-1330; 1972.* (4 references)

Total resmethrin was determined in test formulations prepared in petroleum distillates and in micronized dust formulations by gas-liquid chromatography. The ratio of the two geometrical isomers was obtained from the nuclear magnetic resonance spectrum of the parent acid after hydrolysis of resmethrin. This method of determining isomer ratios was faster and somewhat more accurate than the infrared method. (Author abstract by permission)

73-0740. Carlstrom, A. A. (Chevron Chem. Co., Richmond, CA 94804). **Gas chromatographic determination of phosphamidon insecticide in formulations.** *J. Ass. Offic. Anal. Chem. 55(6): 1331-1335; 1972.* (30 references)

The GLC method described is specific for measuring four components of Phosphamidon Insecticide—deschlorophosphamidon, α-phosphamidon, β-phosphamidon, and γ-chlorophosphamidon. The sample is extracted with acetone, an internal standard (dicyclohexyl phthalate) is added, and the solution is diluted to volume and injected into a gas chromatograph equipped with a flame ionization detector. Although the GLC peak areas for phosphamidon are not linear over a wide range of concentration, good results are obtained when sample and standard concentrations are matched to within 10%. A 1.21% coefficient of variation was found for 19 repetitive injections. (Author abstract by permission)

73-0741. Bazzi, B.; Galluzzi, G.; Abruzzese, L. (Div. Ric. e Brevetti, Ist. di Ric. Agr., Montedison S.p.A., Milan, Italy). **Gas chromatographic determination of *O,O*-diethyl *S*-(*N*-isopropylcarbamoylmethyl) phosphorothiolothionate (prothoate) in technical products and liquid formulations.** *J. Ass. Offic. Anal. Chem. 55(6): 1336-1338; 1972.* (6 references)

A gas chromatographic method has been developed for the analysis of *O,O*-diethyl *S*-(*N*-isopropylcarbamoylmethyl) phosphorothiolothionate (prothoate). The acaricide or a convenient amount of an acaricidal preparation is dissolved in xylene, together with malathion as an internal standard, and analyzed by gas chromatography, using a thermal conductivity detector. The precision ($P = 0.05$) of the method is ±1.5% of the concentration level. (Author abstract by permission)

73-0742. Williams, D. T.; Blanchfield, B. J. (Food Div., Food and Drug Directorate, Dept. of Nat. Health and Welfare, Ottawa, Ontario, Canada K1A L2). **An improved screening method for chlorodibenzo-*p*-dioxins.** *J. Ass. Offic. Anal. Chem. 55(6): 1358-1359; 1972.*

2,3,7,8-Tetrachlorodibenzo-*p*-dioxin is detected at a level as low as 0.05 ppm in corn oil by conversion to octachlorodibenzo-*p*-dioxin and measurement of the latter compound by electron capture gas-liquid chromatography. The method presented is based on a previously reported screening method, which has been modified to include different chlorination conditions and a subsequent alumina column cleanup.

73-0743. Murphy, P. G. (Hopkins Marine Sta., Stanford Univ., Pacific Grove, CA 93950). **Sulfuric acid for the cleanup of animal tissues for analysis of acid-stable chlorinated hydrocarbon residues.** *J. Ass. Offic. Anal. Chem. 55(6): 1360-1362; 1972.* (8 references)

Biological substances interfering with pesticide residue analysis are removed from hexane extracts of animal tissues by shaking them with concentrated sulfuric acid. Recoveries are reported for several chlorinated hydrocarbon pesticides and polychlorinated biphenyls (PCB) from fish extracts. This treatment destroys dieldrin and organophosphate pesticides, but affords a rapid, efficient, and economical cleanup of animal tissues for the analysis of DDT, PCB, and other acid-stable chlorinated hydrocarbon residues. (Author abstract by permission)

73-0744. Doi, Y. (Sect. of Identification, Okayama Prefect. Police Headquarters, Okayama, Japan). **[Enzymatic detection of organophosphorus pesticides using rabbit-liver esterase in thin-layer chromatography.]** *Kagaku Keisatsu Kenkyusho Hokoku (Rep. Nat. Res. Inst. Police Sci.) 25(2): 130-134; 1972.* (9 references) (Japanese)

A thin-layer chromatographic, enzymatic method was developed for detection of ten organophosphate pesticides: methylparathion, papthion (phenthoate), estox, diazinon, fenitrothion, malathion, metasystox (methyl demeton), lebaycid (fenthion), dichlorvos, and trichlorfon. Solutions containing 2-5 μg of the pesticide are placed on the chromatoplates (silica gel layer, 250 μm thick) and developed upward with *n*-hexane: acetone (7:3) or petroleum ether-acetone (9:1) solutions. The layers are air-dried and exposed to bromine vapor for 1 min, then treated with a rabbit liver esterase solution for ca. 60 min at room temperature. The plates were sprayed with a solution of substrate (naphthyl or indoxyl acetate) and diazo reagent, and each pesticide was observed as white spots on a colored background (blue-purple or orange-red). Rabbit liver extract prepared with cold water was more sensitive for detection than the other animal liver extracts. Microgram quantities of the pesticides could be identified using 2-naphthyl acetate as the substrate and Fast Blue B salt as the diazotizing reagent. Other pesticides, including organochlorines and carbamates, were examined for ability to interfere with this reaction. Only captan and chlorfenson showed weak rabbit esterase-inhibiting activity.

73-0745. Biros, F. J.; Dougherty, R. C.; Dalton, J. (Perrine Primate Lab., Environ. Protect. Agency, Perrine, FL 33157). **Positive chemical ionization mass spectra of polycyclic chlorinated pesticides.** *Org. Mass Spectrom. 6(11): 1161-1169; 1972.* (18 references)

The methane chemical ionization (CI) mass spectra were examined for aldrin, isodrin, dieldrin, endrin, heptachlor and its epoxide, nonachlor, α-chlordane, γ-chlordane, and 1-hydroxychlordane. The spectra were all considerably less complex than their electron-impact counterparts. Protonated molecular species of measurable intensity were observed in all oxygenated molecules and those containing an unsubstituted double bond. The fully substituted double bond is apparently a relatively unimportant protonation site. In each case except heptachlor epoxide $[M\text{-}Cl]^+$ was the most intense fragment ion. Retro Diels-Alder fragments were observed for α-and γ-chlordane, aldrin, isodrin, nonachlor, and heptachlor epoxide. The preliminary observations discussed indicate that CI mass spectrometry could be useful in improving analysis of polycyclic chlorinated pesticides.

73-0746. Dougherty, R. C.; Dalton, J.; Biros, F. J. (Dept. of Chem., Florida State Univ., Tallahassee, FL 32306). **Negative chemical ionization mass spectra of polycyclic chlorinated insecticides.** *Org. Mass Spectrom. 6(11): 1171-1181; 1972.* (14 references)

Polycyclic chlorinated pesticides are particularly well-suited for analysis by negative chemical ionization (NCI) mass spectrometry because the electronegativity of chlorine tends to stabilize any of a number of anions which can be formed. The NCI spectra were obtained for aldrin, isodrin, dieldrin, endrin, heptachlor, heptachlor epoxide, nonachlor, α-chlordane, γ-chlordane, and 1-hydroxychlordene using methane, isobutane, or methylene chloride at approximately 0.75 Torr pressure as enhancement gases. The low mass regions of all spectra showed the presence of high concentrations of negative even electron species corresponding to Cl^-, Cl_2^-, H_2OCl^-, and HCl_2^-. Extensive ion molecule association reactions were observed. The base peak in all spectra was associated with the isotopic group of the $[M+Cl]^-$ ion if contributions from other negative even electron ions of low mass values present in high concentrations are neglected. Very little fragmentation was observed. The spectra were reproducible, intense, and interpretable in terms of classical carbanion chemistry. Such data indicate that NCI under the experimental conditions used may be an important new technique for confirmation of certain pesticides.

73-0747. Kirchhoff, J. (Univ. Hohenheim, Lehrstuhl fuer Phytopathol. und Pflanzenschutz, Hohenheim, Germany). **Specific thin-layer chromatographic detection of Gusathion residues in plant extracts.** *Pflanzenschutz-Nachr. 23(4): 355-356; 1970.* (1 reference)

The colorimetric analysis for residues of azinphosethyl and Gusathion (azinphosmethyl) and their oxygen analogs in plant extracts was adapted to identification of the residues on TLC plates. Acetone solutions of extracted material were spotted on silica gel plates. Plates were developed with petroleum ether/chloroform/methanol and sprayed first with *N*-(1-naphthyl)–ethylene diammonium dichloride (0.5% in 20% acetic acid) and then with 6*N* HCl. After heating the plates to 100°, active ingredients turned violet. Plant constituents did not interfere with the detection of 0.1 ppm of the active ingredients or their oxygen analogs, nor did the more than 40 insecticides, fungicides, and herbicides tested.

73-0748. Holz, D. D.; Mayer, Jr., F. L.; Tindle, R. C. (Bur. of Sport Fish. and Wildl., Fish Pestic. Res. Lab., Columbia, MO 65201). **A core-type sampler for pesticide studies.** *Progr. Fish Cult. 34(2): 117-118; 1972.*

An inexpensive core-type sampler was devised which allows a number of sediment samples to be collected and frozen for pesticide analysis. A coring tube was made by grinding the end of a 2 inch PVC pipe to about a 30° angle. The tube was fitted into a reducer which was modified to allow water to escape and to prevent disturbance of the sample, and a handle was provided. After a sample was taken, a new tube was introduced into the apparatus. Use of the sampler is limited by the depth of the water (or handle length) and the consistency of the lowermost part of the substrate. Sand and gravel cannot be sampled with the sampler.

73-0749. Miyamoto, Y.; Onishi, M.; Shimano, N.; Uete, T. (Med. Res. Lab., Kitano Hosp., Osaka, Japan). **[A new method of cholinesterase determination using 2,2′- or 4,4′-dithiodipyridine. I. Kinetic method.]** *Rinsho Byori (Jap. J. Clin. Pathol.) 20(9): 614; 1972.* (Japanese)

A new method for quantitative determination of serum ChE activity was developed. The hydrogen of the SH group of thiocholine formed during hydrolysis of acetyl- or butyrylthiocholine by ChE forms a bond with the nitrogen of the pyridine nucleus of 2,2′- or 4,4′-dithiodipyridine. The tautomeric thio form has an absorption maximum at 343 or 324 nm, respectively. The procedure is as follows: 1 ml of 0.2 m*M* 2,2′-dithiodipyridine (dissolved in 0.2 *M* phosphate buffer solution, pH 7.4) is added to 1 ml of 0.05 *M* acetyl- or butyrylthiocholine iodide, then 0.01 ml of serum added. The extinction coefficient of the mixture is determined periodically at 25°C over a certain time interval. A calibration curve is prepared using thiocholine, and the amount of thiocholine liberated by the unknown is calculated. A linear kinetic relationship is found in this manner between the quantity of serum or the reaction time and the ChE activity. The method appears to be quite sensitive.

73-0750. Onishi, M.; Miyamoto, Y.; Shimano, N.; Uete, T. (Med. Res. Lab., Kitano Hosp., Osaka, Japan). **[A new method for determination of cholinesterase using 2,2′- or 4,4′-dithiodipyridine. II. Non-kinetic method.]** *Rinsho Byori (Jap. J. Clin. Pathol.) 20(9): 614-615; 1972.* (Japanese)

In contrast to the kinetic method described in the previous report, (Abstract No. 73-0749) in which serum ChE activity is measured by determining the thiocholine liberated as a function of time, the present method is based on stopping the reaction after a predetermined incubation time. The procedure followed is similar to that in the previous report, and sodium fluoride is used as the stopping reagent. The serum ChE activity values obtained according to the UV extinction in the present method are in good agreement with those from the ΔpH and DTNB methods except at very low concentrations. In the ΔpH method, the enzyme reaction approaches a limit asymptotically, whereas in the present method and the DTNB method the reaction proceeds in linear fashion in proportion to the quantity of enzyme present. The present method is more sensitive than the DTNB method, and the color developed is more stable.

73-0751. Boler, J. B. (Sentralinstitutt for Industriell Forskning, Oslo, Norway). **Organisk massespektrometri. I praktisk anvendelse pa forskjellige felter. Mulighet for absolutt strukturidentifisering. [Organic mass spectrometry: practical applications in various fields; possibility of absolute structural identification.]** *Tidsskr. Kjemi Bergv. Met. 32(7): 20-23; 1972.* (Norwegian)

Practical applications of mass spectrometry in the qualitative and quantitative determination and the absolute structural identification of organic compounds are outlined. A combination of gas chromatography with mass spectrometry is the best method for the determination and structural identification of very small quantities of organic compounds. This technique is suitable for the determination of PCB in the presence of other pesticides or phthalates, and of DDE and methylmercury. Mass spectrometry can be also combined with thin-layer chromatography.

73-0752. Domanski, J. J.; Sheets, T. J.; Nelson, L. A. (Pesticide Residue Res. Lab., N. Carolina State Univ., Raleigh, NC). **Evaluation of four analytical methods for determining TDE and DDT in tobacco.** *Tob. Sci. 16: 157-159; 1972.* (9 references)

Samples of cigarette tobacco were extracted by four different methods, cleaned up on a Florisil column, and analyzed by gas chromatography to establish the

efficiency of each method in determining residues of *p,p'*-TDEE, *o,p'*-TDE, *p,p'*-TDE, *p,p'*-DDE, *o,p'*-DDT and *p,p'*-DDT. The acetonitrile-water procedure, which involved 10 min soaking with aqueous acetonitrile solution, blending with Hy Flo Super Cel, filtering, and extracting into petroleum ether, yielded significantly greater total TDE plus DDT than the other methods. Low recoveries were obtained with *n*-hexane extraction and benzene soxhlet extraction. The chloroform-methanol soxhlet method was satisfactory and comparable in cost to the acetonitrile-water method, but required an overnight extraction in contrast to a 1 hr extraction. Total TDE plus DDT values obtained by the four methods ranged from 23.06 ppm to 33.64 ppm.

73-0753. Talanov, G. A.; Tsaregorodtseva, G. N. (All-Union Sci. Res. Inst. of Vet. Hyg., Moscow, USSR). **Posmertnaya diagnostika otravleniy zhivotnykh fosfororganicheskimi i karbamatnymi insektitsidami. [Postmortem diagnosis of organophosphorus and carbamate insecticide poisonings in animals.]** *Veterinariya (Moscow) 49(5): 83-85; 1972.* (Russian)

A histochemical method for the postmortem diagnosis of organophosphorus and carbamate insecticide poisoning in animals, based on the detection of cholinesterase, is described. The liver, muscle, brain, or kidney samples to be investigated under microscope are treated with acetylthiocholine chloride, incubated for 2-3 hrs at 37°C, treated for 1-2 min with sodium sulfate solution containing copper sulfate, ammonium sulfide, and finally treated with alcohol. The acetylthiocholine is decomposed by cholinesterase in copper ion-saturated solution into acetic acid and thiocholine which later forms a salt with copper ions. This copper salt then forms a brown, amorphous precipitate of copper sulfide in the presence of ammonium sulfide.

See also **73-0543** **73-0554** **73-0588**

Use of funds
for printing this publication
approved by
the Director of the Office of Management and Budget
February 6, 1973

ORDER FORM
FOR HAPAB

To: Superintendent of Documents
Government Printing Office
Washington, D.C. 20402

Please enter my subscription for Health Aspects of Pesticides Abstract Bulletin. I am enclosing Money Order ☐ Check ☐ for this subscription. ($11.50 a year; $3.00 additional for foreign mailing.)

Please address the HAPAB as follows: _____

_____

_____

UNITED STATES GOVERNMENT PRINTING OFFICE, WASHINGTON, D.C. 1968
For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402. Subscription price $11.50 a year, $3.00 additional for foreign mailing. Price for a single copy of this issue is $1.00, Index issue $2.25.

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS
Technical Services Division
4770 Buford Hwy., Bld. 29
Chamblee, Georgia 30341

POSTAGE AND FEES PAID
ENVIRONMENTAL
PROTECTION AGENCY
EPA-335

If you do not desire to continue receiving this publication, please CHECK HERE ☐; cut off this label and return it to the above address. Your name will then be removed from the mailing list.